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SURGICAL CONSIDERATIONS IN INFLAMMATORY DISEASES OF THE BRAIN AND ITS COVERINGS.[1]

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IN a paper such as this, on surgical considerations in inflammatory diseases of the brain and its coverings, it is impossible to go as much into detail as one would wish. Therefore I trust I will be excused if I brush over some aspects unduly lightly.

In the treatment of intracranial complications much can be done in the way of prevention by the application of care and good judgement to the original condition.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 24, 1931.

One can, for instance, pay great attention to our landmarks in ethmoidal surgery,(1) one can be especially careful of the upper part of the nasal fossæ when patients have had fractures of the anterior fossa, even many years previously,(2) or when the cribriform plates are unusually low or wide.(3) One can avoid performing Sluder's operation on nasal polypi, for in this operation the mortality is five times as great and the recurrence rate the same as by the ordinary methods.(4) One can avoid any instruments which pull or drag when one is resecting the upper part of the septum, and when fractures of the anterior fossa are suspected we can avoid nasal douches.(5) And when the patient is conscious, we can advise him not to blow his nose.(6)

Similarly, in frontal sinus surgery one can carry out such precautions as to avoid radical operations for a few days except in the fulminating cases; one can take care to remove the lining membrane with

gauze swabbing or blunt dissectors and be careful to exenterate the anterior ethmoid cells at the same time, leaving plenty of external drainage. One should be conservative up to a point in frontal sinus surgery and not unnecessarily expose thick cancellous bone to the risk of osteomyelitis. Before performing an intranasal frontal sinus operation one should have a lateral skiagram to show abnormalities in the depth of the cribriform plate, and one should avoid such operations in children under the age of twelve[7] and when other operations in the upper half of the nasal fossa have been performed in the previous three weeks. In fractures of the posterior wall of the frontal sinus one can make an opening about one centimetre in diameter, clean out the clot and use drainage.[8]

In sphenoidal surgery one can take care to observe one's landmarks in sinus punctures and operations[9][10] and always have a lateral skiagram to show which cases have such small sinuses that the risks are out of proportion to the benefits obtainable.

In aural surgery we can avoid unnecessarily early interference with the mastoid in *otitis media.* Before operating we can examine our patients very fully, and whenever the temperature has exceeded 38·9° C. (102° F.) or when even only one rigor has occurred, we can insist on a lumbar puncture and a blood culture being performed. So especially necessary is it not to overlook a labyrinthitis that special consideration will be given to this later. Should an operation be performed, it should be as complete as possible and, if necessary, the dura of both fossæ and the lateral sinus should be inspected. Care must be taken to avoid dislocation of the stapes. Unnecessary shocks from the mallet can be avoided by using Ballance's gouges, where hand pressure alone is used, or machine burrs. A simpler method is to bite off the mastoid tip with *rongeur* forceps and from this beginning to remove the mastoid cortex with the same forceps used obliquely. Generally, curettes and punches such as Kerrison's will then complete the operation without the use of a mallet.[11]

Careful notes of the condition of the mastoid found may prove invaluable in helping one to diagnose the site of intracranial lesions should they develop later.[12]

When fractures of the skull involve the external auditory meatus, it is the consensus of opinion that an antiseptic such as tincture of iodine decreases the risks.[5] If, in spite of this, a mastoiditis develops, a simple mastoidectomy with precautions against jarring should be performed.

The form of labyrinthitis which exerts a threat of intracranial complications is always a diffuse one, and as the linings of the labyrinth and cochlea are continuous, is always associated with a non-functioning cochlea. If, therefore, in a case of mastoiditis with the other ear healthy, the patient hears a fork placed on the forehead loudest in the deaf ear, one can exclude a labyrinthitis causing intracranial complications in that ear. On the other hand, if the fork is heard loudest in the good ear, one must carry out the labyrinth tests. With an unconscious or lightly anæsthetized patient an ocular deviation as the result of spraying the ear with ethyl chloride can still be obtainable with a functioning labyrinth. A totally deaf ear is very suggestive of a possible labyrinthitis. To those who are not aurists, I would point out that total unilateral deafness is easily missed if precautions are not taken to block out the hearing in the opposite ear with a noise-producing machine.

Meningitis.

When Operation is Worth While.

Operation is useless in tuberculous meningitis and well nigh useless in pneumococcal meningitis.[13][14][15][16]

Patients with cerebro-spinal meningitis recover without operation, but the majority of those who die also have sphenoidal sinusitis[17] and some patients do badly until these sinuses are operated upon.

Apart from the cerebro-spinal meningitis, if an organism can be grown from the cerebro-spinal fluid, the prognosis is so unfavourable that, if one knew this condition were present, operation would be hardly worth while. One cannot, however, delay long enough to wait for a culture to grow.

Finding an organism present in a smear from the cerebro-spinal fluid is not of quite such serious importance, as the organisms so found are often dead.

All patients with meningitis of sphenoidal origin, with the exception of cerebro-spinal meningitis, die.[18]

A definite lowering of the chloride estimation in the cerebro-spinal fluid is a sure indication that the meningitis is generalized. The estimation can be done rapidly, and is the means of avoiding operation in hopeless cases. One concludes, therefore, that all patients are worth operating upon, with the exception of those suffering from the following conditions: (*a*) tuberculous meningitis, (*b*) meningitis of sphenoidal origin, (*c*) those conditions in which, when first seen, it is known that organisms from the cerebro-spinal fluid will grow in culture, and (*d*) conditions accompanied by a very low cerebro-spinal fluid chloride content.

Results of Treatment.

Until recently the results of the treatment of meningitis as seen in oto-rhino-laryngological practice have been worse than discouraging. Stewart,[19] reviewing 114 cases in the practice of the Edinburgh Infirmary Ear, Nose and Throat Clinic, 1907 to 1927, found a mortality of 93%.

Compared with such statistics, which were representative for this period, some of the modern results are astounding. Eagleton and Bisset,[20] with 107 cases, had a mortality of only 40%. Watson-Williams[23] had three recoveries in seven cases, whilst Harper[22] had seven recoveries in fourteen cases.

The reason for this great improvement is not only that the principles underlying treatment are better understood, but that diagnosis is made earlier. The classical symptoms of well developed meningitis are very largely death symptoms. Patients with cranial infective disease who are not doing well, should be subjected frequently to lumbar puncture in order that the diagnosis can be made at a time when the best chance of a cure exists, and before any other diagnostic symptoms have arisen.

Surgical Treatment of Meningitis.

Surgical treatment of meningitis can be carried out under the following headings: (i) Removal of site of infection, (ii) drainage, (iii) lavage of meninges, (iv) control of intracranial pressure, (v) keeping the parts at rest, (vi) antiseptics, (vii) use of antibodies, (*a*) passive, (*b*) active.

(i) *Dealing with the Site of Infection.*—Dealing with the site of infection will and has resulted in cures in the earlier stages of meningitis without other treatment, but in meningitis due to chronic aural lesions at least one school has proved to its own satisfaction that better results are obtained by leaving the infective condition and any coexisting intracranial complication, such as a cerebral abscess, until the meningitis is under control.[20]

(ii) *Drainage.*—Drainage to remove organisms and the products of infection should be instituted at or close to the site of infection. Such drainage, if instituted elsewhere, may for a time help to keep down the general intracranial pressure, but will in the end defeat its aim by diffusing infection.

As an example may be cited a case which I once had the opportunity of seeing as a resident medical officer.

The patient, C.M., a male, aged nineteen years, was suffering from chronic mastoiditis, a dead left labyrinth, and meningitis with a high cell count. Operation was considered futile, but the patient lived for thirty-four days. At the end of this time a radical mastoid operation was performed. At this operation the lateral extension of the *cisterna pontis* should have been drained (*viâ* the labyrinth if possible), as a dead labyrinth should have been taken as presumptive evidence that infection had passed up the internal auditory meatus. However, by an error of judgement, the subdural space of the middle fossa was drained, and the patient died in three days. *Post mortem* examination revealed pus tracking up the internal auditory canal and showed that, until recently, the infection was localized to the adjacent parts of the *cisterna pontis*.

Similarly, a case was published of a patient with Gradenigo's syndrome and a definite meningitis who lived for five weeks. Such a syndrome was presumptive evidence that the meningitis started near the tip of the petrous portion of the temporal bone, and that it should have been attacked from the middle fossa. By an error of judgement the *cisterna magna* was drained, and this patient also died in a day or two.

Most of the brain is covered by a shallow subarachnoid space, but the subarachnoid space of the base, starting from that area surrounding the olfactory bulbs to the great cisterns of the posterior fossa, is deep.

Infection of these shallow areas tends to be localized longer, but drainage is more difficult to institute. Dural incision, even with the insertion of suture material as a drain, may not be followed by the flow of any cerebro-spinal fluid. This state of affairs may be due to subarachnoid adhesions, or as Neumann[23] suggests, to an associated encephalitis. Drainage is difficult to institute over the vault or in the middle fossa when this is attacked by the aural route. Here, when no fluid can be obtained, a flow can often be obtained by incising the dura nearer the mid-line.

Shallow subarachnoid spaces exist behind the frontal sinuses and over the orbits. Practically a cistern surrounds the olfactory bulbs. This explains why frontal sinus complications develop comparatively slowly, whilst cribriform plate lesions are followed by fulminating meningitis.[13]

Aural infection, when it spreads to the middle fossa (usually by a small venous thrombosis or by necrosis) tends to be localized for some time and gives rise to meningitis, often associated with symptoms of irritation of branches of the fifth nerve. Gradenigo's syndrome, although arising from a lesion at the petrous tip, also spreads as meningitis to the middle fossa. Cases characterized by this syndrome are usually chronic and usually clear with local mastoid surgical operation.

When aural infections spread backwards, they can spread backwards in a variety of ways. When infection comes from the labyrinth, extension nearly always passes up the internal auditory meatus, and reaches the interpeduncular and pontine cisterns. Drainage can generally be instituted by translabyrinthine drainage up the internal auditory meatus, but when this fails to tap fluid, the lateral extension of the *cisterna pontis* can be tapped just lateral to the internal auditory meatus. It is much rarer for infection to pass from the labyrinth *viâ* the saccus or for necrosis of the posterior semicircular canal to occur. Infection can also reach the meninges of the posterior fossa by direct spread from the mastoid. When the infection passes anterior to the lateral sinus, the treatment is similar, but when it passes posterior to the lateral sinus, it is the *cisterna magna* which is infected. The meningitis from this type is fulminant. As it is stated[12] that cure will not result here unless the treatment is carried out before meningitis is revealed by lumbar puncture, one can see that cures are unlikely to occur. When infection spreads from the lateral sinus, it can spread in various directions, and often the *cisterna magna* is involved similarly.

When drainage cannot be instituted at the site of infection, the nearest part of the adjacent cistern is opened and several strands of silkworm gut are inserted. It is most important that the immediate flow should be free. A small dural incision is less likely to become blocked and less likely to give rise to cerebral herniation than a large one. At any time the flow may cease, but if the symptoms do not recur after twenty-four hours of drainage, this may be looked upon as satisfactory. Patients, however,

have become worse after the drainage has ceased and have ultimately recovered after the second or third attempt at reinstitution of the drainage.

Lumbar puncture in meningitis is only to be mentioned to be condemned, except when small quantities only are removed.[24][25] Certainly patients have been saved by repeated lumbar puncture alone. In these cases the intracranial pressure could have been kept down by other means, which do not incur the same risk of spreading the infection.

Lumbar puncture is necessary for diagnosis, and in these cases not more than six cubic centimetres should be removed at a time.

(iii) *Lavage of Meninges.*—Several workers have advocated lavage of the meninges. To avoid dissemination of infection, the flow must be instituted from a distant part to the site of infection. Normal saline solution cannot be used, for without the addition of calcium and potassium, saline solution is very toxic to cerebral tissue, and a modification of Ringer's or Locke's solution is used. Great care is necessary in the technique, and good results have been obtained, though probably most benefit has been due to the local drainage. One disadvantage of this treatment is that those patients who die in spite of it, often do not have periods of unconsciousness or stupor, but remain conscious in the greatest suffering to the end.[26] The general tendency has been to give up this form of treatment.

(iv) *Control of Cerebro-Spinal Fluid Pressure.*—Naturally, local drainage will control cerebro-spinal fluid pressure, but when the pressure remains high, it can be controlled somewhat by intravenous hypertonic saline solution injections. These lower the pressure chiefly by the diminution they produce in the size of the brain,[27] and this shrinkage will also help the drainage elsewhere. Magnesium sulphate enemata also reduce intracranial pressure.

(v) *Keeping the Parts at Rest.*—Eagleton advocates ligature of the internal carotid when the meningitis is above the *tentorium cerebelli.*[28] The mortality from ligature alone of the internal carotid is very high, being 21% to 45%,[29] but he minimizes this by constricting the vessel slowly with a strip of *fascia lata* under local anæsthesia. If any symptoms of numbness, loss of consciousness, ischæmia of retinæ *et cetera* develop, he releases the constriction at once. When one considers the enormous area of vessels pulsating in the skull, it does not appear worth while to run such risks simply to prevent one internal carotid from pulsating, though such treatment is reasonable in cavernous sinus thrombosis.

(vi) *Antiseptics.*—As elsewhere, antiseptics capable of dealing with the infection are also strong enough to destroy the tissues of the patient. Urotropine is a time-honoured drug in meningitis, but there is no evidence that it does any good. Jenkins has elaborated a complicated technique for drainage and lavage in meningitis of aural origin.[24] He used a specially prepared suspension of iodoform in modified Locke's solution. Although Layton[25] describes Jenkins's results as brilliant, time will show whether the iodoform used in the technique takes a part in the results.

(vii) *Production of Immune Bodies.*—It has been shown that, although the meninges possess powers of resistance against infection, antibodies are not normally present in the cerebro-spinal fluid.

Some authorities have injected intramuscularly the patients with infected cerebro-spinal fluid with the idea of producing an active immunity, but such immunity would probably protect only the brain itself.

Others have advocated massive lumbar puncture with the hope that antibodies might be so induced to leak into the cerebro-spinal fluid. Such punctures, however, tend to spread meningitis, and experiments in animals with a bacteriæmia show that in them a massive lumbar puncture will actually produce a meningitis.[30]

Injection of antibodies into the theca often causes severe reactions with a great rise of intracranial pressure, but when local drainage is satisfactory, probably little, if any, harm could accrue from their use, though they would soon be washed away. It has been shown that in experimental pneumococcal meningitis in animals injection of antibody does not cure the animal, though it prolongs its life.[31]

Cerebral Abscess.

Cerebral abscesses arise as the result of: (i) direct spread of infection, (ii) retrograde thrombosis of veins, (iii) an infected embolus.

Therefore, the first two occur as the result of adjacent infection, though in retrograde thrombosis of veins there may be a considerable layer of healthy brain intervening. In an infected embolus the infection is from a distance, and the abscesses are liable to be multiple. Some of the abscesses previously regarded as hæmatogenous, as in bronchiectasis, are now known to belong to the former group of infection and are due to spread from the usually coexisting sinusitis.

Treatment.

Treatment consists of: (i) Waiting until the preceding encephalitis has broken down into pus. In some cases of suspected abscess, pus never develops, and in many of these cases the true pathological condition is that of a non-infected cerebral infarct. (ii) Locating the pus with a needle. (iii) Draining the abscess. (iv) In the case of cerebellar abscess, taking precautions that the intracranial pressure of the cerebellar fossa does not become excessive.

In the Mayo Clinic[32] it was found that the best cases to treat were those in which the symptoms were indistinguishable from symptoms of a cerebral tumour.

In evolving the technique for any case it must be remembered that drainage of an abscess (unless sterile, as it often is) through healthy brain may

cause spread of the abscess or meningitis. On the other hand, exploration of healthy brain through an infected area will almost certainly cause encephalitis or meningitis.

At the same time it must be remembered that the greater number of recoveries from cerebral abscess occur in those cases in which an abscess is drained through the stalk connecting it to an infected area.

In the various sites these difficulties are best met with as follows.

Temporo-Sphenoidal Abscess.—In treating a temporo-sphenoidal abscess the operation is as follows:

An inverted U-shaped incision is made over the ear of the affected side. This is carried to the bone and the pinna is displaced downwards, though the cartilaginous meatus is not detached. An inverted J-shaped incision is made above this, with the short limb of the J anterior and the flap turned downwards and forwards. The middle fossa is penetrated with a burr or trephine and an opening about 3·1 centimetres (one and a quarter inches) in diameter is made. Sometimes the abscess is subdural, when it tends to track upwards in this direction. If the dura appears abnormal, it is opened with a small incision, and if subdural pus is found, bone is removed to the full extent of the abscess and, after the dura has been incised to the limits of the abscess and a drain is put in place, the ear is dealt with. If, however, no pus is found, the dura of the middle fossa is elevated until the area of the *tegmen tympani* and *tegmen antri* is exposed. If the dura is here abnormal, it is reasonable to assume that a stalk is present and the wound is closed, and further treatment and exploration are carried out through a mastoid operation. If, however, the dura appears normal, the temporo-sphenoidal lobe is explored with a wide bore needle on a syringe, a stilette being used to clear the needle. In order to minimize the risks of herniation, no incision is made in the dura, and, should an abscess be tapped, to avoid the risk of infection spreading back along the tract and then up the previously unsuccessfully needled tracts, each exploration is made through a separate dural puncture. If pus is struck, a fine bore rubber catheter impaled on a stilette is passed alongside the needle into the abscess. After a few days larger catheters are inserted from time to time. If the abscess was due to aural disease, the mastoid is dealt with at the end of the operation, but before the mastoid incision is made, the previous incisions are tightly closed and the area where the mastoid incision joins on to Lake's incision is packed with gauze soaked in 2% iodine solution to promote rapid adhesions and diminish the risk of secondary infection from the ear.

If no sign of dural involvement has been found and needling has been unsuccessful, the wound can be closed without any risk of having infected the brain.

The method advised carries with it little shock in contrast to the formidable operation of Eagleton. By exposing the brain first, one avoids the necessity of changing towels, instruments *et cetera* in the middle of the operation, and one has the absolute certainty that the field is clean. Puncture of the dura without incising it first was advocated by Lund.[33] Kerrison[34] has laid stress on using separate puncture sites for each exploration, and Lund has done most to popularize Lemaître's method of gradual dilatation of the drainage tract that has given most favourable results. Coleman[35][36] has been most successful with Lemaître's method of drainage and he had nine consecutive recoveries in cerebral abscesses. Later, his series enlarged to twenty cerebral and four cerebellar abscesses. Of the twenty-four patients, seventeen with cerebral and two with cerebellar abscess recovered.

Cerebellar Abscess.

When a cerebral abscess is suspested and a dead labyrinth is present, it is presumptive evidence that the abscess is cerebellar.

Owing to early pressure on the aqueduct of Sylvius, the intracranial pressure is high, and if the dura anywhere posterior to the anterior vertical part of the lateral sinus is incised, the cerebellar substance will often exude like the contents out of a tube of tooth paste.

In cerebellar tumour operative surgery, the mortality was high until it was found that this pressure must be first dealt with. This was done by removing all bone from one lateral sinus to its fellow of the opposite side and from the superior curved line down to the *foramen magnum*. If the pressure was still high, the lateral ventricle was tapped also.

It is only recently that the same principle has been applied to the surgery of abscesses in this region, with the happy results that Eagleton and Bissett report a recovery rate of 50%.[20]

When the dura in front of the lateral sinus is opened, there is not, for some reason not apparent, the same tendency for herniation to occur. This is the area first to explore, as abscesses are twice as common in front of the lateral sinus as behind it.

After the decompression operation has been completed, it is decided whether to go on with the operation or to wait a day or so for the patient to recuperate from the effects of the decompression operation and from the internal hydrocephalus.

The mastoid operation is then performed, including a labyrinthectomy, if a dead labyrinth exists, and the dura exposed in front of the lateral sinus. If an extradural abscess is found, exploration should not be proceeded with until the effects of drainage have been observed, although often a coexisting intradural collection of pus exists also. Intradural exploration should not be carried out except where one is very certain that a cerebellar abscess is present. This is because exploration will be through an infected area, and, in any case, after a thorough mastoidectomy the wound should be syringed with 2% iodine before the exploration is carried out. If pus is found, drainage is again established by Lund's method, and it is important to allow the pus to escape slowly.[37]

In cases of cerebellar abscess associated with a thrombosis of the lateral sinus, the exploration can be carried out through the median wall of the sinus after the sinus operation has been completed.

Frontal Abscess.

Abscesses in the frontal area are less frequently diagnosed before death, as definitely diagnostic symptoms do not arise until the abscess bursts into the ventricle or the motor area is pressed upon. This accounts for the high mortality.[38] As much as 240 cubic centimetres (eight fluid ounces) has been found at operation[39] in these cases, and death usually occurs suddenly whilst the patient is still active.[40]

In connexion with this complication in frontal sinus cases, the author has always found a decreased or absent homolateral abdominal reflex. The phenomenon has also been present in severe frontal

sinusitis when recovery has occurred without cerebral exploration and when probably there was no more than a congestion of the adjacent brain. He holds that normal abdominal reflexes are almost conclusive evidence that complications have not taken place in frontal sinusitis.

These abscesses arise by: (i) direct spread from the frontal or ethmoidal sinuses or overlying bone, (ii) retrograde venous thrombosis, (iii) metastasis.

In metastasis, trauma is not an uncommon concomitant cause. Eagleton[41] states that the frontal lobes are more or less fixed by small veins which pass to the meninges, and he adds he has never seen a fractured skull at autopsy in which there is not ecchymosis in the frontal lobe as a result of rupture of these vessels.

The frontal lobe is explored through an osteoplastic incision in a clean area above the frontal sinus. This obviates missing a subdural abscess (25% of cases[30]), as one is likely to do through a small incision. If the dura looks normal, it is stripped back and the dura posterior to the sinus is exposed. If this is inflamed, one can assume that a stalk is present, and the wound is closed and further treatment and exploration are carried out through the frontal sinus.

If the dura behind the posterior surface of the frontal sinus appears normal, needle exploration is carried out as described for temporo-sphenoidal abscess and similar treatment instituted.

Eagleton[42] states that subdural abscess is so frequently associated with intradural abscess in the frontal region that here one must not be content with drainage of an extradural collection, but must carry out exploration also for an intradural collection.

When frontal abscess is secondary to osteomyelitis of the frontal bone, no treatment will effect a cure, for in spite of anything that may be done for the abscess, the osteomyelitis will produce a fatal result.

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A MODERN CONCEPTION OF MENINGEAL INFLAMMATION.[1]

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MAN'S supremacy in the vertebrate kingdom is consequent upon the specialization and sensitivity of that vast and intricate collection of constantly vibrating fibres which we call the central nervous system. Its problems and mysteries have absorbed the attention of the anatomist, the physiologist, the surgeon and the physician from the earliest days.

The finer methods of histological technique have revealed many secrets of pathological structure

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 29, 1931.

which help to elucidate functional disorders in the cells and tracts of the nervous tissues themselves, but of Nature's method of protecting and conserving those functions, of the mechanism of a fluid cushion confined within membranes structurally unique, and of a system of pressures constantly balanced, comparatively little was known until recent years.

In 1914 Harvey Cushing wrote:[1]

> Our knowledge of the meningeal and ependymal coverings of the central nervous system, as well as of the part played by the fluid which circulates through and over them, has hardly kept pace with our knowledge of the nervous tissues which they enwrap. This is the more remarkable when the recent invasion of the cerebro-spinal space for experimental, diagnostic, therapeutic and operative procedures is taken into account. As regards the cerebro-spinal fluid we stand in much the same position as did the pre-Harveian phlebotomist in regard to the circulation of the blood.

Let us consider the development of the membranes and the circulation of the fluid.

In the study of embryos it has been shown that the cerebro-spinal space is formed by means of fusion of clefts between mesodermal crests about the neural plate.[2] As the nervous tissue differentiates, in the region of the fourth ventricle there is a condensation of tissue which forms the transverse part of the chorioid plexus, and a dorsal evagination from this is cut off and permits the escape of fluid into the surrounding tissues. These are mesodermal cells at first arranged as a syncytium, the inner layer of which becomes intimately associated with the developing brain to form the *pia mater*; the intercellular strands form trabeculæ to connect with the outer layer or arachnoid membrane. The external layer of the mesoderm here undergoes a fibrous thickening, first in the region of the base of the skull, and the *dura mater* develops after the subarachnoid space has become outlined.

The functional difference between the *dura mater* and the pia-arachnoid has been the basis of a series of experiments by Burr and Harvey,[3] who believe that certain ectodermal cells of the neural crest are essential for the formation of the lepto-meninges. They found that in embryonic transplants of cerebral tissue without neural crest cells, a fibrous covering analogous to *dura mater* was formed, whereas if portion of ectoderm were included, a villous structure resembling the chorioid plexus, fluid spaces and a vascular network developed.

So we see, as the nervous system develops, the enveloping fluid layer keeps pace, the foramen of Magendie is early formed to permit the escape of fluid secreted by the chorioid plexus, and the path of the future circulation is established.

The mechanism of secretion of the fluid is by the specialized cells of the chorioid plexus, the vascular, ependymal covered fringes hanging into the lateral ventricles of the brain. This organ of secretion is postulated to behave as a dialyser, the fluid passing out from the blood, modified by certain selective powers of the secreting cells, and by the general conditions of intracranial pressures, distension of cerebral veins, variations in arterial pressure and respiration.

The fluid passes out into the subarachnoid space through the foramina in the roof of the fourth ventricle, up over the surface of the cerebral hemispheres and down into the spinal canal, where it is absorbed by the arachnoidal villi of the cerebral sinuses. Weed and his coworkers[4] have made use of the Prussian blue reaction to demonstrate the mode of absorption. They injected isotonic solutions containing potassium ferrocyanide and iron ammonium citrate into the cerebro-spinal canal under normal pressures, and subsequently precipitated the salts as Prussian blue at the point of absorption.

By this means the cell condensations of the arachnoid which project into the cerebral sinuses, particularly along the superior longitudinal sinus and the transverse sinus, were demonstrated to be the site of absorption of the greater part of the fluid secreted by the plexus. It has also been shown[7] that in the spinal canal there is an additional mechanism of arachnoidal cell clusters in the perivascular spaces along the emergence of the spinal nerves at the reflection of the membranes. Where Prussian blue is deposited, in artificially produced cerebral anæmia, when the intracranial pressure is markedly lowered and the ferrocyanide introduced, these spaces are found widely dilated and distended with fluid: it is suggested that this may be a reversible phenomenon and the fluid pressures thus preserved.

Similarly, intravenous injections of hypertonic saline solution will dilate the spaces and relieve conditions of increased intracranial tension.

Hence there is a balanced mechanism for secretion and absorption to maintain the physiological equilibrium of the pressure system within the closed box of the cranio-spinal cavity,[6] consisting of the central secreting chorioid gland, the apertures to permit escape of fluid, its circulation in the intra-lepto-meningeal space, and accessory channels for its escape in conditions of altered secretion or vascular supply.

Now what is the nature of this fluid? We have said it is a dialysate from the blood and shows clear evidence of the remarkable selective action of the plexus, for it contains some substances in greater concentration than is found in the blood, some in less; and there is an absolute barrier action of the plexus against foreign substances injected into the blood stream and against protective substances formed in the blood in response to certain infections.

The actual amount of fluid present under normal conditions is probably about 130 cubic centimetres, and undergoes slight changes during its circulation and absorption. The ventricular fluid is almost free from cells, and its protein content is also appreciably lower than that of the lumbar fluid. Whether any secretory substance is added to the fluid by the hypophysis or any excretory products

are passed out, are questions almost impossible to answer.

Thus we obtain a clear picture of the plexus, fluid and membranes as a preliminary to a discussion of their response to infection, that is, the inflammatory reaction exhibited by this type of surface membrane, for such we must consider it; and here, as in all surface membranes, there are two modes of production of inflammation, one by local irritants, one by agents circulating in the blood stream.

Local inflammations are usually dependent upon trauma or focal conditions in adjacent organs—the pachymeningitis beneath a fracture of the cranial vault or in relation to intranasal suppuration or middle ear disease. Here the first effect is upon the *dura mater*, which reacts as does any connective tissue, and its inflammation cannot affect the cerebro-spinal fluid. Should, however, the condition involve the arachnoid beneath, there may be a local shedding of cells—the chemical constitution of the fluid will be unaltered, as indeed will be its amount, unless altered conditions of intracranial pressure have stimulated the plexus. Mildly irritant fluids may also be brought into contact with the membranes through the medium of the lumbar puncture needle. Highly albuminous fluids, such as normal horse serum or the various antitoxic sera, may produce a cellular reaction from the arachnoid and increase the permeability of the plexus in the maintenance of the colloid balance, and produce symptoms of the condition called serous meningitis. Under these circumstances the glucose content of the fluid remains unaltered, and is a useful guide where a suspected bacterial infection has been anticipated by serum injections, and the cell content of later fluids may give rise to some doubt as to the cause of the reaction.

The response of the chorio-ependymal layers to blood-borne infection, however, is a much more complex process, and its study presents many problems difficult of solution. The barrier action of the plexus has already been mentioned, and it is upon the factors which, by their assembled forces, are able to break down that barrier, that the development of meningitis depends.

Let us here comment upon the term "meningitis." Actual inflammatory change is in the great majority of cases confined to the chorio-ependymal layers. It is here that the cellular exudate is poured out; it is here that the invading organism breaks through and finds in the cerebro-spinal fluid an autogenous culture medium awaiting its multiplication, and so alters the permeability of the plexus that we find gross degrees of alteration in the fluid.

One of the earliest changes demonstrable in the fluid is the acquisition of a substance normal to the blood, namely, complement. Greenfield[5] has utilized the hæmolytic system indicator to demonstrate its presence before protein content or cell count has risen to give the danger signal. The next change is in the salinity of the fluid, a falling chloride content often being found in a fluid released under increased pressure and with a cell count below double figures. This leads us to the condition so familiar to the pædiatrician—meningismus. A child suffering from an acute infection—pneumonia, pertussis, gastro-enteritis — exhibits marked irritability when handled, holds its head back in the pillow and has a Kernig's sign. Lumbar puncture is performed and thirty cubic centimetres of clear fluid under a pressure of 240 millimetres of mercury are removed, the condition is relieved and further puncture is unnecessary. The bacteriæmia which can frequently be proved to exist in these cases, has resulted in the lodgement of some organism in the meshes of the chorioid plexus whose barrier action is called into play; and while the stimulus for increased formation of fluid has shown in the increased pressure, the subsequent relief proves its success.

This conception naturally leads on to the more severe infection, where organisms are more virulent and actively multiplying in the blood, or perhaps have a selective affinity for the meninges. Here the barrier breaks, and the plexus, functionally impaired, pours forth a fluid of altered chemical composition and marked cellular increase, this last being the main factor in the difficulty of treatment. As the inflammatory cells accumulate and clog the filter of the arachnoid villi, which are designed to deal only with fluids, the cells poured out to aid by their phagocytic action the fight against the invading organism thus defeat the purpose by their physical presence. They accumulate in the most distensible spaces, that is, where the venous pressure is lowest, and along the areas of rapid absorption of the fluid, that is, along the basal cisterns, the Sylvian fissures, the frontal and parietal lobes. This distribution of pus is a commonplace at autopsy.

The development of agglutinins and specific antibodies by the cells of the plexus, or their passage from the blood, is very small; the direction of specialization of the reaction seems to be towards cell production and phagocytosis and autogenous lavage by excessive secretion. It has been well demonstrated by Stewart[9] that in laboratory animals infection introduced into the ventricles of the brain can be accomplished by a dose of only a fraction of that necessary by intravenous injection. The cerebro-spinal canal constitutes a non-immune locus, and when once established, the microorganism has every facility for a winning battle, and it is in recognition of this fact that we hold as the greatest weapon in our armoury against these micro-organisms the immune serum.

Forms of Meningitis.

Epidemic Cerebro-Spinal Meningitis.

Epidemic cerebro-spinal meningitis, spotted fever, is from the prognostic point of view the mildest infection with which the meninges have to deal. During the Great War epidemics occurred in various training camps in England as well as in the fighting zone, and full advantage was taken of the oppor-

tunity for investigation and improvement in methods of treatment.

The meningococcus is one of a large group of Gram-negative cocci frequently found in the naso-pharynx of otherwise healthy people, and on human passage, such as occurs in crowded communities, may become enhanced in virulence and, multiplying in the blood, gain access to the meninges, giving rise to typical cerebro-spinal fever. Its identification in the non-contact population is difficult, and routine swabbings in a number of investigations have given surprisingly high figures. But as Arkwright says:

> It must be borne in mind that no really final answer can be given to the question whether a given Gram-negative coccus is truly an infective meningococcus or not, unless it comes from the meninges.[10]

Diagnosis by cultural methods needs to be controlled by agglutination tests. A polyvalent rabbit serum appears to be the best for this purpose.

The experience gained in routine search for this organism reveals its incidence to be as much as 12% to 15%, even in the absence of clinical cases, and this figure again to be dependent on overcrowding; when the rate under these circumstances rises to 20% cases begin to occur. Gordon describes this as the danger level of meningococcus pressure in a given community.

The organism is considered of low virulence as regards the blood and respiratory tract, but of marked virulence towards the meninges, hence investigations of the bacterial metabolism have been made in an effort to discover the reason. In addition to the reductase enzyme there is an endotoxin which is heat-stable and water soluble. This is used in the identification of Gordon's[10] serological Types I and II which form the largest proportion of his series of 526 cases—37·66% and 44·05% respectively. Pursuing this fact, monovalent sera were used, their potency depending on the proportion of anti-endotoxin produced in the test animal. Type I infections were the most active in the production of effective antiserum, and in the later years of the Great War amongst soldiers treated with these sera the mortality was 35% compared with 60% in the civil population to whom polyvalent serum only was available.

Reference has been made to the low virulence of the meningococcus in the respiratory tract and blood stream. This is of significance in the mechanism of production of skin petechial and purpuric spots.[11] The organisms deposited here from the blood stream cause an immediate reaction and are overcome, the metastasis in the chorioid plexus finds its selected spot, persisting and multiplying in the ventricular fluid, but once overtaken by the inflammatory cell it readily undergoes phagocytosis. The realization of this blood infection has led to the trial of intensive intravenous serum therapy, doses of 100 to 200 cubic centimetres being administered. Banks[12] reports a series of 15 patients so treated, with nine deaths; six of those who died were under five years of age. One male patient, aged eighteen years, had but one dose of serum, 200 cubic centimetres given intravenously on the third day of illness, and was discharged cured after four weeks. He records complete absence of serum reactions in these cases. This is a truly remarkable instance. The permeability of the chorioid plexus to such a high concentration of immune bodies in the blood was unfortunately not tested, so that the efficiency of the method has but for its proof the patient's recovery.

Pneumococcal Meningitis.

With the pneumococcus we have a similar range of serological types of organism, a similar possibility of blood infection from a respiratory tract focus, with the difference that the virulence of the organism has usually manifested itself locally before dissemination, as in middle ear disease and in suppuration of the cranial air sinuses, and that the leucocytosis of the blood and the permeability of the plexus and inflammatory exudate are much greater. Here the organisms, once planted in the cerebro-spinal fluid, find its salinity and sugar content an admirable medium and multiply with extraordinary rapidity. A high antibody content is soon reached in the blood, but no agglutinins are found in the fluid.

Stewart,[9] in an admirably controlled series of experiments, demonstrated in both rabbits and dogs that with a blood serum agglutination titre of 1:20 intraventricular inoculation of small amounts of S forms of Type II pneumococci would produce meningitis, while enormous intravenous doses would fail to produce it. Methods of treatment were then investigated, lavage being the first, cisternal and lumbar puncture being supplemented by occasional ventricular lavage through a trephine opening; but if complete washing of the ventricles was not attained, residual organisms would bring about reinfection leading to the animal's death. Pneumococcal antiserum combined with lavage, methylene blue in saline solution, mercurochrome,[14] "Optochin"[13] (ethyl hydrocuprein hydrochloride) have all been used in an effort to free the masses of inflammatory cells. Monovalent sera, however potent, must in this case be assisted by mechanical lavage. The small numbers of recorded recoveries, and the varying substances used in all instances, would indicate that success is due to the common factor of lavage rather than to any individual merit of the various antiseptics.

Influenzal Meningitis.

Influenzal meningitis is becoming more important, at least in this city. At the Royal Alexandra Hospital for Children we have seen six cases in the past three months, all terminating fatally. There is apparently no relation between the meningeal infection and influenza epidemics. The cases appear to arise spontaneously, with little or no antecedent respiratory infection.

The influenzal organism is a delicate one, with a distinct predilection for the meninges and nervous tissues; psychoses are of frequent occurrence as a

sequel to some forms of the respiratory type of infection. Greenfield[15] reports a case of disseminated encephalomyelitis of influenzal origin.

Animals used for experiments, with the exception of the white mouse, are resistant to infection, and rabbits treated with increasing doses of respiratory strains of the organism produce no antibodies and react but slowly to meningeal strains.[16] The organism may grow in symbiosis; we have seen a case in which Pfeiffer's bacillus was associated with a pneumococcus; the latter died out in the third culture, and at autopsy a pure culture of *Bacillus influenzæ* was obtained. This form of infection is even more fatal than the pneumococcal, the average duration of life being from six to eight days.

Marcus and Crane[17] consider this also a primary blood infection and record positive blood cultures.

In the one recovery I have been able to find reported convalescent serum from a patient with respiratory infection was used, combined with free lavage.

Streptococcal Meningitis.

Streptococcal meningitis would appear to be more common in the northern hemisphere than in the southern. The records of the Royal Alexandra Hospital for Children contain but two cases in the past five years, neither of which complicated an ear condition. J. T. Smeall[18] lists the hæmolytic streptococcus as causing 73% of cases with mastoiditis and meningitis at the Royal Infirmary, Edinburgh. The non-hæmolytic variety is less toxic, and of our two patients one lived three months and died of extreme wasting and malnutrition, but with a normal cerebro-spinal fluid, while the other child has made a complete recovery.

Tuberculous Meningitis.

Now let us turn to the more chronic type of meningeal affection, namely, that produced by tuberculosis. The termination of the life of a small child reared in a home where a parent or relative has an open pulmonary lesion, is frequently dramatic. The child, previously well, is noticed to become fretful and disinclined for food; the parents conclude that he is "growing too fast" until suddenly he is found to have a squint, to be extremely drowsy, and rigid in his neck muscles. A brief three weeks of deepening unconsciousness, broken only by the high-pitched meningeal cry, perhaps convulsions, and autopsy reveals the lesions of a widespread tuberculosis affecting abdominal and thoracic organs, brain and lepto-meninges.

How does all this come about? The whole question of infection by a bacillus to which the human race has developed such a high degree of resistance is one of wide range; it calls for a consideration of phenomena in adults which account for a difference in response and termination of infection in childhood, namely, resistance and immunity—the condition by which the tissues and body fluids become unfavourable to the life and growth of the invading organism. Krause has said in regard to tuberculosis that resistance may be produced only by living bacilli, that is, by making the individual tuberculous. In considering the subjects of tuberculous meningitis we are faced with the fact that in all probability these patients are suffering from the effects of a first massive dose, a dose sufficiently large to produce overt infection. With other children the dose may be such that the theory of Krause is fulfilled, that is, immunity may be established. The familiar tuberculin skin test will give a positive result in both. We are using an extract of the protein of the bacillus to test the tissue reaction to that protein; the child who has had a dose of any magnitude will respond with the development of an allergic phenomenon of vaso-dilatation and cell exudation; the tissues are sensitive to the proteins of the bacillus and hasten to combat it. But does this test tell us of the result of that combat? No, it merely tells us of a sensitizing infection. Witness the tissue cultures of sensitized animals placed in tuberculin: they die, while tissues of a normal animal continue to grow.[19] The sensitized animal, when reinfected intratracheally with tubercle bacilli, responds with proliferative inflammation, while a normal animal responds with tubercle formation, and as the bacilli multiply, develops the allergic state parallel with the attempt at immunity; as phagocytosis goes on there is liberated the lipoid substance from the bacilli which stimulates tubercle formation, and so the cycle continues.

Resistance, then, will be the balance between the allergic reaction and the neutralization of the products of that reaction.

So it is uncommon to find in these children a single lesion of any great size to which one may point as a primary focus, for that fact of itself would indicate the development of resistance; one finds perhaps a pulmonary lesion one or two centimetres in diameter with no actual caseation, but which has permitted the bacilli to multiply in such numbers that, when the necrotizing substance produced in the disintegration of some of them has exerted its influence on a blood vessel of any size, there is produced a systemic infection in which tubercle formation is the rule, that is, the allergic response is less prominent in this showering of one or two bacilli into the capillaries of the spleen, liver, lung and brain, for this is the stage of tubercle formation. Throughout the viscera tubercle formation is usually subperitoneal and of necessarily limited spread, but in the brain it is a somewhat different story, on account of the circulating fluid without the pial membrane. Here it is possible for the bacillary products, and finally the bacilli themselves, to gain access to the fluid; for it is an experimental fact that the barrier action of the plexus is preserved even more efficiently in tuberculosis than in acute infections; relatively enormous doses, producing a fatal tuberculosis in animals, will not infect the meninges. In confirmation of this it is possible to demonstrate in an extraordinarily high percentage of cases of terminal

meningitis a focus on the surface of the brain; indeed, in one case we have demonstrated tubercle formation in the chorioid plexus itself.

The realization of this method of production of tuberculous meningitis also provides us with an explanation of the occasional cures seen in cases typical of the disease. Is it not possible that in a cerebral lesion which has discharged into the cerebro-spinal fluid a certain amount of toxin to produce an inflammatory reaction causing symptoms of meningitis, there may form arachnoidal adhesions around the lesion sufficient to prevent the generalization of the organism and permitting the tubercle to engulf the bacilli even as a calcifying gland engulfs them?

These cases, I think, must be regarded as having successfully concluded the struggle to establish a resistant state, while the products of allergic inflammation have contributed very largely to the fierceness of the battle, if not to the ultimate victory.

So, in brief review of the subject of meningeal inflammation in the light of modern research, we realize again the perfect protective mechanism of the chorioid plexus and the circulation and absorption of the cerebro-spinal fluid in regard to the nervous tissues. We realize the massing of defences against invading forces tending to break down that mechanism, and the difficulties in combating infection, even as the post-Harveian physician, appreciating the significance of the discovery of the circulation of the blood, realizes more fully the problems yet to be solved.

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SOME THOUGHTS ON THE TERM ACUTE DISSEMINATED ENCEPHALOMYELITIS AND ITS AFFINITIES.[1]

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ANY discussion on the inflammatory phenomena which may be associated with the central nervous system would hardly be complete without including some reference to the state called acute disseminated encephalomyelitis or what it practically commonly amounts to, post-vaccinal encephalitis. The more common infective states of the central nervous system fall naturally to the pathologists of general hospitals, but the affinities of the above condition, acute multiple sclerosis on the one hand and *encephalitis lethargica* and general paralysis on the other, bring it nearer to the ordinary activities of the neurological laboratory attached to mental hospitals. This is my excuse for bringing this state before you, which, as you are aware, has of late been given much prominence in the old land. Again, owing no doubt to its association with the public health aspect of vaccination, an emotional effect has crept in, and some issues have been confused.

Has the Condition a Specific Ætiology?

Some would include a special cellular pathology, making much for a double origin for the infiltrating cells. Thus a mesoblastic reaction would include endothelial and plasma cells, while neurotropic viruses exciting an ectoblastic phenomenon would include the glia, called microglia. But these we now know can become *Gitterzellen*, a part of the reticulo-endothelial system. Moreover, astrocytes can accompany many bacterial infections as well as *encephalitis lethargica* and general paralysis; and Orr and Rows long ago described as polyblasts derived from lymphocytes near vessels in their injected nerves many of the particular forms of cells dilated on by present day workers in post-vaccinal encephalitis. Others have shown that similar pictures of demyelination can occur after any of the exanthemata or even after obscure infections or no infections at all. Still further controversy is concerned with the question of whether one virus is responsible for encephalomyelitis, no matter in what sickness it

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 24, 1931.

appears or whether any germ or virus which can cause the exanthemata *et cetera*, can also, if it attacks the central nervous system, give rise to this state, the host being weakened first of all by his special disease.

The special virus incriminated may be that of herpes, or *encephalitis lethargica*, or one postulated. Armchair critics point out its seasonal incidence is not that of *encephalitis lethargica*, and lymph harmless in Madrid may be associated with post-vaccinal encephalitis in Holland. And again they state that, while the pathological picture of measles encephalitis exactly resembles the post-vaccinal type, yet its morbidity is much less, and so the same virus can scarcely cause both. It has even been asked whether the type with demyelination has the same ætiology as that in which demyelination is not present. While discussing the various ætiologies put forth and different aspects or stages of the histological pictures outlined, I shall seize opportunities of comparing these pictures with those obtaining in many other infective states in the central nervous system and incidentally shall uphold Kinnier Wilson among others who point out how similarly the brain reacts, no matter what the trauma, and also how widespread the lesions are.

Typical Lesions in Encephalomyelitis.

Nervous symptoms showing up about ten days after vaccination commonly have the following pathology: (i) Congestion and hæmorrhage, (ii) perivascular mononuclear infiltration, which may be remarkably slight, and have (as some maintain) specific features of its own, and (iii) usually, but not necessarily, a more outlying perivascular demyelination, affecting both grey and white matter. The pathological picture naturally will vary with the stage at which death takes place.

Demyelination, then, in large areas is a striking but not essential accompaniment of the encephalitis and/or the myelitis. This is important. Usually in sections of spinal cord stained after the Pal-Weigert method there may be seen areas of perivascular demyelination so numerous as to appear bilaterally symmetrical. According to Turnbull, lesions may be obtained from cortex to lumbar levels. The lesions also follow veins and their branches for long distances. Innumerable perivascular and peri-adventitial small and large lymphocytes, plasmacoid and true plasma cells abound, and the demyelination may also be marginal and in the peripheral nerves. Demyelination in each lesion is usually complete, except round the edges, and only a fraction of the axis cylinders survives. Perivascular glia forms an abnormally close network, although partly by imbibition rather than by hyperplasia. Later one gets a vast mosaic of transformed microglia, that is, *Gitterzellen* which infiltrate the tissues widely. The changes in the glia are obviously important in comparing the condition with disseminated sclerosis. Vascular adventitial sheaths not only contain cells but coagulated albumin. Turnbull thinks that these histological appearances differentiate the lesion from that of poliomyelitis and *encephalitis lethargica*. Neurones are often astonishingly well preserved. Neuronophagia may be absent. Commonly one finds many signs of lepto-meningitis and encephalitis. Turnbull would group this disease with Westphil's disseminated sclerosis and Schilder's disease.

Other Infectious Diseases with Similar Lesions.

Others deny the specificity of these lesions in the infection of vaccination, and Wohlwill found the same state apart from any known infection at all. Redlich's case more resembled disseminated sclerosis. Wohlwill also describes cases after measles, but with less meningeal and perivascular reaction. There is no doubt now that encephalomyelitis can occur in the course of smallpox, measles, mumps, chickenpox, antirabic treatment, and other less defined illnesses, and even be reproduced thence in animals. With vaccine material Hurst and Fairbrother readily induced an infection in the brains of rabbits and monkeys which they stress was primarily meningeal. Because of this they would entirely differentiate it from post-vaccinal encephalitis of man with its rich histopathological changes. They would also sharply differentiate it from polioencephalitis which, having ectotropic properties, calls forth an ectodermal response, including certain glial components, while the vaccinal virus in their animals produced an essentially mesodermal reaction. From this they argue that vaccine virus is not the cause of post-vaccinal encephalitis. MacIntosh and Scarff quote Levaditi and Nicolau and others to show that with exalted vaccine or other virus a true mononuclear meningomyelitis is readily obtained; and, using a highly diluted vaccine virus themselves, they avoided the acute hæmorrhage reaction of the acute form (as is also typical of acute *encephalitis lethargica*), and instead produced more chronic lesions in their animals quite comparable to those of post-vaccinal encephalitis. They support Ledingham, who showed the essential lesion was a granuloma of the small blood vessels in the spleen *et cetera*.

The mesoblastic reaction included an outgrowth of endothelial cells from the intima and adventitia, resulting in a diffuse perivascular and periadventitial infiltration of cells and a true encephalitic reaction as well as cells invading from the pia, a real activity of the reticulo-endothelial system. This means that they claim to find in the brains of laboratory animals the same lesion as in post-vaccinal states. They do not consider the glaring demyelination to be the essential lesion, since this is found in the central nervous system in injuries, post-vaccinal encephalitis, subacute combined degenerations, disseminated sclerosis, cerebral syphilis and measles. They suggest, therefore, that viruses and infective agents which can produce the exanthem, can also, when they attack the nervous system, produce demyelination; that is, they have this power in common.

Post-Influenzal Types.

Greenfield has pictured *post mortem* two cases of encephalomyelitis following influenza, which, on naked eye and histological examination, almost exactly duplicate the post-vaccinal type. One case was acute and the other of five weeks' duration, and thus two stages could be compared. Greenfield reasons from all this that some common virus must be responsible for encephalomyelitis. Most authorities agree that demyelination proceeds much faster than in Wallerian degeneration, yet multiple softening is quite a misnomer; no extreme colloquative necrosis of the area takes place as in septic conditions or hæmorrhage. Care must be taken to distinguish infiltration of the pia by cells draining an area of demyelination from tabetic conditions in which all the pia may be deeply infiltrated. Many epidemics of influenza are complicated by venous thrombosis and naturally the brain can also be similarly involved.

The Encephalitic Element in Encephalomyelitis.

Although in encephalomyelitis the neurones commonly escape, yet at times nerve cell destruction in cord stem or brain may be a feature, and in this respect the condition has affinities with many other forms of encephalitis. We know that in very acute cases of *encephalitis lethargica* the cortical cells escape remarkably, and our attention is attracted by the number of mononuclear cells round them or along capillaries, and especially by changes in the vessels and by mononuclear meningitis with hæmorrhages while as yet glial reaction is indefinite. Later on we see neurone destruction and proliferation of astrocytes. In acute infective polyneuritis in soldiers we see an anterior poliomyelitis and encephalitis together with aggregations of mononuclear cells in lungs, liver, kidneys *et cetera*, and globoid bodies may be recognized in cultures.

Somewhat similar lesions in the anterior horns have been described in "pink" disease, but I fancy with Dr. Wilfred Evans that "pink disease" is probably only a syndrome causable by several agents. I have so far found inflammatory changes (and slight ones at that) in only one out of seven cases, and the cord changes in "pink disease" suggesting combined sclerosis, pointed out by American writers, can possibly be explained by delayed myelination in those under twelve months. Critchley states that in Parkinson's paralysis "no part of the central nervous system is immune, for changes occurred throughout the cerebrum, cerebellum, spinal cord and even neuromuscular junction." Ganglion cells were diminished and unhealthy in *globus pallidus*, mid-brain, *substantia nigra*, and there was destruction of myelin and overgrowth of glia.

In Landry's paralysis a form of poliomyelitis is not uncommon. However, one of my cases included a marginal myelitis as well. In Sydenham's chorea, in addition to numerous small hæmorrhages in the occipital lobe, we see elsewhere many of the changes characteristic of *encephalitis lethargica*. Embolism of some of the smaller blood vessels may cause some of the more permanent lesions. Chorea is therefore a meningomyelitis of rheumatic origin, wherein vascular changes prevail. In Poland, in a severe epidemic in children resembling encephalitis, Flatau found cellular infiltrations of the basal ganglia, cord and peripheral nerves with glial reactions near the vessels and leptomeningitis; less commonly he found neuronophagia. He would have encephalomyelitis an aftermath of *encephalitis lethargica*. Knauer and Jaensch maintain that the cerebrospinal fluid often harbours the virus of *encephalitis lethargica*, and by slit lamp methods thought that they could show constant keratitis in laboratory animals. From this he maintains that the encephalitis following measles, whooping cough, influenza and vaccination must be due to some special virus resistant to cold *et cetera* and which must accompany all these diseases and be the chief causal factor in the encephalomyelitides. He also maintains that this virus is that of *encephalitis lethargica* and that bacteria cannot cause this particular keratitis.

Affinities with Disseminated Sclerosis.

Briefly, we may consider disseminated sclerosis as including large and small areas of sclerosis with more or less demyelinations in both grey and white matter in brain and cord, which areas do not pick out definite tracts, do not run up long distances in the affected areas, that is, do not give rise to ascending and descending degenerations, mainly preserve their axis cylinders intact, include the richest possible network of glial fibres, are commonly not so numerous or large as the corresponding areas in encephalomyelitis, do not show the same cellular infiltrations, but do present the same rich mosaic of *Gitterzellen*. Recent ideas include the chronic and subacute as one disease fading into one another, though the acute type seems nearer to encephalomyelitis. Vascular connexions are not always clear, but if they are involved, the lesion usually includes but one segment of vessels only and not their branches. One view is to consider the process as related to a non-purulent encephalomyelitis allied to poliomyelitis, rabies *et cetera;* and Dawson holds the view that it is a subacute encephalomyelitis ending in areas of actual sclerosis. Here at least it seems unnecessary to blame the blood vessels, although I must admit that in my acute cases the sclerosed areas revealed intense fibro-hyaline degeneration of the medium sized vessels and thickened walls and narrowed lumens.

Finally, an English committee on post-vaccinal troubles admits many affinities between subacute disseminated sclerosis and acute disseminated encephalomyelitis, and quote Pette as describing a case "in between."

Observations on Kindred Diseases in Animals.

Conditions allied to multiple sclerosis seem to occur naturally in dogs, and Perdrau and Pugh in natural and experimental distemper found lesions

on the one hand resembling those following measles and vaccination, and on the other perhaps more inclining to disseminated sclerosis. Tracey Putnam, while experimenting on dogs with injections of tetanus toxins, also noted an acute multiple sclerosis in a few cases—discrete patches of demyelination with perivascular and meningeal infiltrations and gliosis. These findings seem illuminating in view of the work of Eckel and Winkelman on "productive arteritis." These workers produced proliferation of the intima of blood vessels in animals by the intravenous injection of weak acids *et cetera*. They noted their likeness to states found in children in connexion with food poisoning and certain obscure infections, and added that there was nothing specific in the productive arteritis found in syphilis—it was a common reaction in a vessel subject to the assault of toxins, spirochætal or chemical. In the cerebellum of these children he found areas of demyelination with intense *Gitterzellen* infiltration and gliosis. I have such a cerebellum from the children's hospital showing obvious productive arteritis and encephalomyelitis with intense *Gitterzellen* infiltration and gliosis; it is in truth a case connecting the two conditions and also, by the way, disclosing a fitting pathology for the "cerebellar" type of encephalomyelitis described by Brain and Strauss. This, too, seems a good place to call in the assistance of the biochemists who dabble in the buffer qualities of the blood *et cetera*.

Neurotoxin or Vascular Obstruction.

At this point it seems feasible to examine evidence which will decide whether the lesions found in all these diseases with so much in common are due to a neurotoxin or to malnutrition from vascular states or from both. The case for primarily a pure toxin action seems proved for at least one group of cases, for Weston Hurst, by inoculating anterior poliomyelitic material into the sciatic nerve of an animal, showed that the anterior horn cells revealed degenerative changes before any other tissue reactions; later on, of course, other infiltrations came about. I had long thought I could ascribe the same properties to the *Spirochæta pallida*, and it was therefore with surprise that I read Bruce Hawes as follows:

> The *Treponema pallidum* is an organism which throughout the centuries has almost learnt to live in symbiosis with man. Although possibly emitting, containing or manufacturing some toxin, this toxin has usually no very virulent and rapid effects on the tissues.

And again:

> The changes that are present in nearly all types of syphilis are vascular changes—the spirochætes prefer the inner coats of the blood vessels. These vessels develop narrowed lumens (throw out new capillaries) and thus give rise to nutritional changes and fibrous overgrowth *et cetera*.

With so many capillaries involved we should expect a slowly evolving condition of undernutrition in the nervous system, and this he maintains is true for tabes *et cetera*, and goes on to point out that office workers get tabes while coolies with overworked anterior horns and so on get syphilitic myelitis and Erb's syphilitic spinal paralysis and even amyotrophy. This latter condition is not unknown in *encephalitis lethargica*. I think those enterprising workers Orr and Rows at first thought in terms of toxin when, after placing germ-filled celloidin capsules next the sciatic nerves of rabbits, they noted an interstitial reaction in dura and pia-arachnoid, round cell infiltration and degeneration in the posterior nerve roots, root zones and posterior columns (a reaction they called lymphogenous). After placing similar capsules next to intra-abdominal sympathetic ganglia or intestines, they noted very little inflammatory reaction near the cord, but islands of demyelination in the posterior and lateral tracts. This state so much resembling the combined scleroses after pernicious anæmia they named "hæmatogenous." In 1931, however, one of them, Orr, now showed that the lesion at the site of the capsule was interstitial in character, and, coming to brain lesions, contended that the richness or otherwise of the arteriolar anastomoses had a bearing on the extent of the lesions.

Tabetic Lesions: Some Recent Views.

In regard to tabetic lesions, Hassin writes:

> Degenerative changes in posterior columns and arachnoid parts of roots are a primary process due to disturbed circulation of the tissue fluids in the spinal cord. The "inflammatory" changes occur in the dura and arachnoid, in which they invoke inflammatory phenomena. In the dura they occur as infiltrations of hematogenous elements and vascular changes typical of syphilis; in the arachnoid as proliferations of arachnoid cells (Richter's "granulation cells") which invade perineural spaces and obstruct them, interfering with the cerebro-spinal fluid flow. From this stasis we get rarefaction of the spinal cord tissue showing as islands of myelin degeneration in the posterior columns.

Hassin thus holds with the epidural origin of tabes.

Ruby Stern, from Queen's Square, while noting these views, inclines to the Orr-Rows ideas, which include toxins from the hosts of spirochætes in the aorta reaching the cord or at least the dura by way of the peripheral nerves *et cetera*. These workers at least suggest how some forms of demyelination can come about. Finally, not all deaths after vaccination are due to calf lymph. Temple Grey and Whittaker report two cases thus diagnosed in which the lesions, multiple brain softenings, were due to a staphylococcal and some other bacterial effect, and quote Diamond, who writes:

> In septicæmia one finds no organism in the brain, though histological examination may reveal multiple areas of softenings due to toxin reaction and not definite germ infiltration.

He compares these lesions to those occurring in disseminated sclerosis. No disease must be labelled "encephalomyelitis" until septic states be excluded.

Finally, boils and injuries about the vertebral column may lead to patches of demyelination called "compression myelitis" with changes in the neurones and the new facts in connexion with intracranial pressure reveal a histology disconcerting to the pathologist bereft of an adequate history.

We may truly agree with Temple Grey that there is a need for a histological survey of the central nervous system in common hospital ailments so that suitable controls will be always available in the laboratory.

Summary.

It would seem that the striking syndrome of many islets of demyelination in cord and brain as seen in post-vaccinal encephalomyelitis reveals not a disease of specific entity, but a form of lesion which can occur (even if rarely) during the course of innumerable other diseases, mostly infections.

Further, whether the causal agent is the germ of the particular exanthem involved or a special virus which creeps in or is inactivated for evil during that exanthem, the rôle of impaired vascular function must not be overlooked.

Finally, the pathology of acute disseminated encephalomyelitis includes phenomena common to many other diseases, and even states induced by chemical means in the central nervous systems of animals.

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GOITRE PROPHYLAXIS.

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GOITRE is very prevalent in certain parts of Australia, such as Gippsland and some areas in Tasmania. That it is common in Melbourne is shown by the fact that during the last five years 260 patients with colloid or adenomatous goître and 270 with exophthalmic goître were admitted to the wards of the Melbourne Hospital, while many others were treated in the out-patient department.

Marine[1] states that of all known diseases simple goître is the easiest and cheapest to prevent. In addition to the control of cretinism and those forms of mutism and feeble-mindedness that are dependent on thyreoid insufficiency, the abolition of simple goître would eliminate adenomatous goître with its frequent complications. Since malignant disease of the thyreoid gland almost always arises in a preexisting adenomatous goître, the incidence of this form of cancer would be greatly reduced.

Other beneficial effects might accrue from the use of the minute amounts of iodine necessary for this purpose. Stevens[2] is confident that senile arterial changes are the result of some defect in iodine metabolism, and considers that the use of iodides may prevent, retard or arrest degenerative arterial changes. Arteriosclerosis is of frequent occurrence in cretinism and in adult myxœdema, and Goldberg and Simpson[3] found that it was common in lambs deprived of their thyreoid glands. It has been stated that carcinomata in general appear earlier in life in highly goîtrous districts than in non-goîtrous areas. The elimination of endemic goître would prevent this form of premature senility.

In endemic areas cretins are liable to be found in the third generation of goîtrous parents. A history of goître in the mother is not infrequent among Victorians presenting themselves with colloid or adenomatous goîtres which sometimes are congenital.

Kimball and Marinus,[4] in a survey of more than 36,000 children in Michigan, found 10% more goîtrous than non-goîtrous children on the delinquent list. Mental retardation was more frequent in children with goître. Among 3,548 backward children in special schools in Detroit, 281 suffered from congenital hypothyreoidism, while a further survey showed that 10% of all feeble-mindedness was the result of endemic goître. Of these 281 persons, 45% showed a falling intelligence quotient or a fixed mental age (cretinoid type of mental growth). In New Zealand the lower mental acuity of children in goîtrous districts has been observed.[5] In Switzerland this type of mental deficiency constitutes a real social problem, as shown by the fact that there are 2,000 cretins in the city of Berne alone.

Obviously the efficient prophylaxis of simple colloid goître has far-reaching effects.

The distribution of iodine in air, water, various soils, plants and animals and its relation to the incidence of goître were first fully investigated by Chatin.[6] His work was amply confirmed by Fellenberg.[7] McClendon and Hathaway[8] demonstrated the relationship between the iodine content in plants and drinking water and the incidence of goître in the United States of America. Hercus, Benson and Carter[9] have shown that in New Zealand the incidence of goître varies inversely with the amount of iodine in the soil which is reflected in the iodine content of the foodstuffs raised on it. Orr,[9] after preliminary investigations of the iodine content of foodstuffs in relation to the incidence of goître in England, concluded that, despite the excellent results obtained in the prophylaxis of goître by iodine, the available evidence was insufficient to determine whether iodine deficiency was a cause of goître. Marine[10] and his coworkers found in various animals that if the iodine content of the thyreoid gland was constantly above 0·1% of its dried weight, goîtrous enlargement did not occur.

Most observers agree that simple goître is apparently always related to iodine deficiency, either an

absolute deficiency of iodine intake in endemic areas or a relative iodine deficiency in relation to the needs of the individual in sporadic cases. The influence of diet, infections and the physiological stresses of puberty, pregnancy and the menopause are important in this connexion.

The prevention of simple goître was successfully attempted by Marine and Kimball[11] in 1917. Two grammes of sodium iodide were given in ten doses during a fortnight in the spring and the autumn to 2,190 schoolgirls, while 2,305 others acted as controls. Over a period of two and a half years five of the former and 495 of the latter group developed goître. Marine[1] quotes numerous Continental observers who have confirmed the value of iodine in the prophylaxis of endemic goître. Kimball and Marinus[4] state that the introduction of prophylactic measures in the Detroit schools reduced the incidence of goître from 42% in 1924 to 7% in 1929. The latter cases were nearly all instances of congenital goître. In their opinion the endemic goître problem in Detroit and Michigan has been solved. Silberschmidt[12] states that among 9,500 children in Zurich the incidence of goître has fallen from 86·4% to 14·7% as the result of treatment for four years with a weekly dose of five milligrammes of iodine.

Before the general use of iodine for the prophylaxis of goître is justified in a population with a goître incidence decidedly lower than in endemic areas, the possibility of ill-effects from this procedure must be considered. That no harm will come to the patient with a normal thyreoid gland from the use of relatively large doses of iodine is demonstrated daily by the therapeutic use of iodine. Likewise in children before the age of puberty it is generally agreed that relatively large doses of iodine can be given with impunity even to those suffering from goître. But in adults with long standing goîtres, the use of iodine in such doses for long periods may be followed by toxic manifestations.

Kimball,[13] among 2,659 patients with thyreotoxic symptoms, found 309 in whom the condition was apparently precipitated or made worse by iodine. Six of these patients over forty years of age who had adenomatous goîtres, had used iodized salt (apparently containing more than one part of potassium iodide to 10,000 parts of salt). The others had all taken iodine in large doses for long periods, in most instances under medical advice. Hartsock[14] in 1926 stated that in at least one-fourth of all patients with hyperthyreoidism operated on at the Cleveland Clinic in the preceding six months the symptoms appeared to have arisen or been aggravated as the result of the use of iodine. Sixteen males, only two of whom were using iodine therapeutically for goître, developed symptoms in from one to eighteen months after starting to use iodized salt of a strength greater than one in 10,000 parts of salt. Eighteen females over middle age with long standing adenomatous goîtres likewise developed toxic manifestations within a year of commencing to use this salt, while in thirty-two others with nodular goîtres an exacerbation of the previously mild toxic symptoms resulted. After using this salt five girls between twelve and eighteen years of age developed symptoms of hyperthyreoidism, while three patients who had been in good health for from four to thirteen years after a thyreoidectomy, suffered from a recurrence of their thyreotoxic condition. During this period iodide rashes were more frequent in the clinic, while patients with *acne vulgaris* were made worse or failed to respond to treatment while iodized salt was being used. Carmalt Jones[15] reports that at one college the use of iodized salt of a strength of one in 5,000 parts of salt was followed by the development of thyreotoxic symptoms in three patients. Hercus[16] regarded the increase in the incidence of toxic goître in New Zealand between 1925 and 1927 as almost certainly due to the unwise use of iodine in goître therapy and possibly in prophylaxis.

Though the long continued use of relatively large doses of iodine may result in the development or exacerbation of toxic symptoms in patients with certain types of goître, all the observers quoted above are enthusiastic advocates of the proper use of iodine in goître prophylaxis. Hotz[17] states that the daily intake of iodine must reach 0·7 milligrammes (700 γ) before any harmful effects of iodine on a pathological thyreoid gland need be anticipated.

It is important to realize the almost infinitesimal amount of iodine necessary for the prophylaxis of goître.

Marine[18] holds that the use of massive doses of iodine was the chief factor in bringing iodine into ill repute in the earlier empiric treatment of goître, and is still the major cause of untoward effects. He states that the maximum storage capacity of the normal human thyreoid gland for iodine is about twenty-five milligrammes. Orr and Leitch[19] found that the average iodine content of the normal thyreoid gland was about eight milligrammes. Fellenberg[7] observed that the average daily intake of iodine in a non-goîtrous area in Switzerland was 31 γ, while in a goîtrous region it was 13 γ. Hercus and Roberts[20] found the average difference in the daily intake of iodine in goîtrous and non-goîtrous districts was 14.7 γ, the average intake in the latter areas being 34·85 γ. The foodstuffs naturally richest in iodine are edible seaweed, oysters, tinned fish, eggs, sea fish, wholemeal products, leafy vegetables and milk in this order. They estimate that the normal human thyreoid requires about 10·4 milligrammes of iodine *per annum*, or about 30 γ (0·03 milligramme) daily. The average daily consumption of salt was estimated at about six grammes.

All non-iodized salts in common use in New Zealand proved to have an excessively low iodine content. The use of iodized salt containing one part of potassium iodide in 250,000 parts of salt would thus supplement the average iodine intake by 24 γ daily or 8·76 milligrammes per year.

It is remarkable that so small an addition to the annual amount of iodine ingested should have such dramatic effects, but the method has proved satisfactory in Switzerland, where Eggenberger[21] estimated that in February, 1924, over 700,000 inhabitants were using iodized salt daily. In February, 1922, the Canton Appenzell am Rhein introduced a standard iodized salt of a strength of one part of potassium iodide to 200,000 parts of salt. Two years later the percentage of babies with congenital goître had dropped from 50% to nil and the number of goître operations had diminished by 75%.

There is no evidence that these minute amounts of iodine have any ill effects, though, as shown previously, the use of salt containing from two hundred to four hundred times this amount of iodine does produce harmful effects in certain cases.

Various methods have been used to supply the general population with this extra iodine. In Rochester, New York, the addition of iodine to the water supply proved wasteful and uncertain. This applies to the use of manures rich in iodine to

increase the iodine content of foodstuffs grown on manured soils, and also to the addition of potassium iodide to the food of cows.

Krauss and Munro[22] showed that from 10% to 15% of the iodine given appeared in the milk.

Jarvis, Clough and Clark[23] state that the systematic use of sea foods rich in iodine would be of benefit in the prevention and treatment of simple goître. They showed that the average iodine content of tinned salmon is nearly one milligramme per kilogram dried weight or 0·3 milligramme per kilogram moist weight. This food is easily available for inland communities. Keith[24] gave an instance of the value of salmon in goître prophylaxis. In the Pemberton Valley in British Columbia every white child was born with goître and most adults developed it soon after settling there. The mortality among their newborn stock varied from 85% to 100%. There was no goître among Red Indians in the valley nor among their stock. They ate a great deal of salmon and annually cured large numbers for winter food, while their pigs consumed the fish washed ashore from the stream.

The advantages of using iodized salt are the low cost and the fact that every individual in the community is reached by this means.

McCollum and Simmonds[25] give an interesting example of the harm that frequently results from our efforts to improve on Nature. In Charleston, Virginia, before 1900 goître was very rare. Soon after this, 60% of schoolgirls in Charleston and Huntington suffered from goître. There had been no change in the general food and water supplies, but in 1898 refined table salt replaced the coarse salt formerly obtained from local wells.

There is fairly general agreement that the best method at present available for the general prophylaxis of goître is the use of iodized salt of a strength approximating to four parts of potassium iodide per million parts of salt.

The objection to the limitation of goître prophylaxis to children up to or just beyond the age of puberty is that it fails to reach the most important class of pregnant women. If the method be applied to the school population only, the best means appears to be the individual weekly administration of one tablet containing five milligrammes of iodine. This method has proved successful in Switzerland[12] and in the United States of America.[4]

Kimball,[20] after years of experience of this method, states that he has never seen an authentic case of hyperthyreoidism resulting in a child from the school prophylaxis of goître. Marine[1] advises the administration of ten milligrammes of iodine every week throughout the period of pregnancy and lactation to all women living in a goîtrous area.

This dosage could probably be reduced by at least one-half without any ill effect and possibly with benefit. Burroughs, Wellcome and Company market a product, "Tabloid Brand Iodicin (chocolate base)" which contains ten milligrammes of iodine in organic combination in each tablet.

In my opinion the goître problem in Victoria and Tasmania (and probably in other parts of Australia) is of sufficient importance to justify the introduction of those general methods of prophylaxis which have proved of value elsewhere. As applied to school children, the method has no harmful effects, nor are any seen with the general use of iodized salt if the proportion of potassium iodide added does not exceed four parts per million of salt.

However, the prophylaxis of goître need not wait upon the activities of the public health authorities. The practitioner can play a most effective part. During pregnancy his patients may be given iodine in any form in doses not exceeding five milligrammes (one-twelfth of a grain) weekly. Children may receive a course of iodine twice a year, as was originally practised by Marine and Kimball.[11] A convenient method is the administration of syrup of hydriodic acid, which contains 1% of iodine, in doses of four mils (one drachm) daily for a fortnight twice a year.

We are all so keen to take advantage of any advance in methods of treatment of established disease that we seldom consider the doubtful value of some of these from the point of view of the race. For example, considering the influence of heredity in such disorders as *diabetes mellitus* and pernicious anæmia, the introduction of insulin and of liver therapy for these diseases is not an unmixed blessing. On the other hand, the prevention of a disease such as goître raises the standard of health of future generations. The practitioner frequently regards the prevention of disease as the work of the health authorities and as something beyond the sphere of his immediate activities. This paper has been written in an effort to stimulate interest in the prevention of a common disorder and to emphasize that much can be done in this way by the individual practitioner.

Conclusions.

1. Of all known diseases, simple goître is the easiest and cheapest to prevent.

2. Its prevention would have beneficial effects on many related disorders.

3. Iodized salt of a strength of one part of potassium iodide in 250,000 parts of salt is an effective and safe prophylactic for general use.

4. If prophylaxis be limited to the school population, the ingestion of five milligrammes of iodine every week is effective and has no harmful effects.

5. The use of prophylactic measures during pregnancy is of the utmost importance.

6. The individual practitioner can play a most effective part in the prophylaxis of goître.

7. Simple goître is, however, sufficiently common in certain parts of Australia to demand that the health authorities should take steps to institute appropriate prophylactic measures.

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Reports of Cases.

DETACHED RETINA CURED BY GONIN'S OPERATION.[1]

By W. LOCKHART GIBSON, M.B., Ch.M. (Sydney), F.R.A.C.S.,

Honorary Ophthalmic Surgeon, Brisbane Hospital.

I FIRST saw this patient at the Brisbane Hospital on July 16, 1931. He was complaining for two weeks of defective vision in the left eye and said that the defect was getting worse. He had been struck on the left eye six weeks previously with a nail.

Examination revealed the vision of the left eye to be $^{6}/_{36}$. Complete loss of the field was present in the lower nasal quadrant. There was a detachment of the retina involving practically the whole upper temporal quadrant. Two small holes were found in the detached part of the retina about ten millimetres from the limbus. The holes were very small and there was just a narrow band between them.

[1] The patient described herein was shown at a meeting of the Queensland Branch of the British Medical Association on September 4, 1931.

Gonin's theory of "spontaneous" detachment of the retina is that it is caused by a hole or holes in the retina which allow fluid from the vitreous to pass through them and so get behind the retina and separate it from the chorioid. The object of the operation is: first, to find the hole or holes in the retina; second, accurately to localize the hole or holes; third, to occlude the hole or holes.

The occluding of the holes is done by reflecting a flap of conjunctiva from over the area where the hole is, then opening the sclera over the hole so that fluid escapes from behind the retina. The point of a white hot cautery is then put into the opening. The operation can be done under local anæsthesia.

It is probably not necessary to put the point of the cautery actually into the hole in the retina, but it should be as near the hole as possible, so that the exudate from the chorioid will stick down the edges of the hole and so prevent fluid passing through it, in the same manner that fluid is prevented from passing through the pupil in iris *bombé*.

The operation to occlude the hole is not difficult, but the finding and localizing of the hole is often very tedious. The operation on this patient was done on July 28 and he now has a full field of vision, sees $^{6}/_{12}$, and there is no detachment. One operation only was done, as the holes were so small and so close together. It is possible that the vision will further improve, as the vitreous is not absolutely clear.

The object of showing this case is to bring before the notice of medical practitioners the fact that many cases of spontaneous detachment of the retina can now be cured and that the earlier they are treated, the better the prognosis. "The Medical Annual" for 1931 states that there are more authentic records of cures of retinal detachments published during the past year than can be put to the credit of all other methods of treatment during the last twenty years.

Acknowledgement.

I am indebted to Dr. George Thomson for kindly checking the localization and assisting at the operation of occluding the holes.

FOREIGN BODIES IN THE RESPIRATORY TRACT: TWO CASES.

By HAMILTON KIRKLAND, M.B., Ch.M. (Sydney),

Aural Surgeon, Sydney Hospital; Assistant Aural Surgeon, Saint Vincent's Hospital.

Case I.

MRS. A.G., aged fifty-three years, consulted me in May, 1930, with regard to a sore throat and odynphagia commencing fourteen days earlier and rapidly growing worse.

There was marked fœtor in the breath. An immediate inspection of any part except the mouth was precluded by a mass projecting from the dorsum of the tongue, a large thyreoglossal cyst which had been present for many years. Under general anæsthetic and with the use of "Brühning's direct vision laryngoscope, the pharynx, larynx *et cetera* were examined, and after considerable search a sloughing mass was discovered at the base of the tongue. Moderate traction dislodged a flat piece of bone some 2·0 by 1·5 centimetres impacted anterior to the epiglottis. A large pressure ulcer had resulted therefrom. Recovery was uneventful and the patient refuses any operation on the cyst.

The interesting points are that the history gave no indication of a foreign body, in spite of close questioning, before or after operation; there was difficulty in obtaining a view of the vallecula on account of the large, firm thyreoglossal cyst lying just anterior to that space.

Case II.

Mrs. C. was sent to me in September, 1930, by Dr. Sinclair, of Nowra. The history was that some two months

before, whilst eating stew, something caught in her throat and for some seconds she was unable to breathe. Then the obstruction freed itself and she had a bad fit of coughing followed by persistent dyspnœa. She came to Sydney and was admitted to a general hospital for investigation. X ray examination of the chest and laryngoscopic examination revealed no abnormality and she was discharged with a diagnosis of asthma.

Later on, following another severe bout of coughing, the dyspnœa became worse. Dr. Sinclair very properly doubted the original diagnosis and sent the patient to me.

On examination, the patient was slightly dyspnœic, but not cyanosed, and a dry rhonchus could be heard all over both lungs. X ray examination again revealed no abnormality, but I considered she probably had a foreign body in the trachea.

Under local anæsthesia a bronchoscopic examination was done and a piece of bone 3·5 by 2·0 centimetres was located at the bifurcation. This was quite easily picked out with Brühning's forceps. Within a few days the patient had made a complete recovery.

It was fortunate in this case that all tissue had been stewed from the bone, or its decomposition would almost certainly have caused a fatal lung sepsis.

Reviews.

LECTURES IN MEDICAL HISTORY.

A WELL-KNOWN Sydney physician once complained that most of his house physicians had no knowledge of anything outside the subjects of their curriculum. To prevent that reproach being merited by future Adelaide graduates, at least as far as a knowledge of the history of their profession is concerned, Dr. Bernard Dawson prepared and delivered to the medical undergraduates a course of lectures on that subject. These have now been published.[1]

We may say at once that this small volume admirably succeeds in fulfilling its author's object. Any student who will take the trouble to read it two or three times should possess a sound framework of medical history, the interstices of which he can fill in with greater detail at his leisure. The space allotted to the different periods is well balanced, and the author, in telling the story of the evolving drama of medicine in an interesting manner, shows a good judgement in his choice of the principal actors in it, and his style avoids the two extremes of prolixity and conciseness.

There are a few minor points on which we wish to comment. We cannot agree that the quotation from the Ebers Papyrus (page 9) shows that the ancient Egyptians recognized the pulse; indeed, Dr. Dawson contradicts himself on this point on page 121. The statement merely refers to the omnipresence of the arteries and does not even imply that they contain blood. Still less does it show a knowledge of the circulation. The absence of syphilis from ancient Egypt is mentioned. It is of interest to recall that the lesions of ancient bones from that country, alleged by French scientists to be of syphilitic origin, were shown by an Australian, Professor G. Elliot Smith, to be due to the ravages of longicorn beetles. Dr. Dawson does not refer to the aid given to the Egyptian study of anatomy by the practice of embalming. He derives the word "chemistry" from "Chemi," the Black Land, the old name of Egypt. That derivation is not universally accepted, but in any case we think him wrong in stating that for that reason chemistry is known as the "black art." This term arose in the middle ages from the popular corruption of "necromancy" into "nigromancy" and had a much wider application than to chemistry. We cannot discover that Aristotle advocated the use of the formal laws of logic in scientific investigations. He certainly developed and classified those laws for a very different purpose, namely, as weapons in the dialectical discussions then so popular among his countrymen. But the object of the disputants was to secure a victory in debate without regard to the truth of the matter debated.

The assertion that the Dorians occupied Crete, Cos and Cnidus obscures the fact that they also possessed a large part of south Greece. It is stated that the Roman Empire was finally divided into eastern and western portions in the sixth century. Gibbon places the final division in the fourth century. Dr. Dawson implies that the Franks occupied Gaul after the death of Attila. The truth is that Gaul was occupied by the Suevi, Alani, Burgundians and Vandals in 405 A.D. and the Franks merely joined them in 420 A.D., whereas Attila did not even invade Italy until more than thirty years later. We regret to find Dr. Dawson repeating the statement that the Roman legions abandoned Britain to the Saxons. In spite of its complete refutation and the exhibition of its curious origin, this absurd error is still copied from history to history. The credit of the introduction of the podalic version is given both to Soranus and to Galen. It is affirmed that syphilis was recognized in Europe in the thirteenth century. It would have been wiser, however, to warn students that many authorities still insist that it was unknown there until it was introduced from America in the fifteenth century. The account of the middle ages might have been illustrated by reference to Kenelm Digby and his ointment of honour. It is disappointing in an English history to find the Americans credited without protest with the discovery of inhalation anæsthesia and no reference made to the pioneer work of our own countrymen, Sir Humphry Davy and Dr. Hickman. While recognizing the limitations of space in such a work, we expected to find some mention of Thales and Roger Bacon, if not of the better known but less important Francis Bacon.

In a subsequent edition we hope that Dr. Dawson will add the dates of birth and death, where known, of every important character mentioned. Without this information the relation of different men to each other is sometimes obscure. We would also urge him to add another chapter dealing with such moderns as Virchow, Ehrlich, Claude Bernard, Brown-Séquard, Langley, Gaskell, Helmholtz, Mackenzie and Hughlings Jackson.

It will be observed that all our criticism is in matters of detail and does not detract from the value of the book. The volume is pleasingly bound and clearly printed on good paper and its illustrations are well chosen and reproduced. It contains few misprints, but "millenia" (page 7) and "entrants" (page 121) require alteration, and "nutritive" (page 126) should surely be "fatty." The first word in the Greek quotation on page 39 lacks its breathing.

We hope that this book will be bought, read and re-read by all medical students in Australia and by many overseas, as well as by those graduates who are still students but who have not yet studied the development of their calling.

[1] "The History of Medicine: A Short Synopsis," by Bernard Dawson, M.D., F.R.C.S.; 1931. London: H. K. Lewis. Crown 8vo., pp. 174, with illustrations. Price: 7s. 6d. net.

ANÆSTHESIA AND ANÆSHTHETICS.

THE second edition of Dr. C. F. Hadfield's "Practical Anæsthetics" is now published in the "Practical Handbook Series" of intermediate text books.[1] In the eight years that have passed since the book first appeared, new drugs have been discovered and there have been many advances in methods. This has necessitated revision of th subject matter and amplification to the extent of another ninety pages. The book is eminently practical and shows throughout the marks of a sound teacher from whose wide experience are gleaned the essentials that must be stressed, and matters unproven or debatable which may be omitted.

As no book can reflect the entire teaching of more than one man, the critic will find points with which he would have dealt differently. The arrangement of the book is peculiar, and some changes might be made for the benefit

[1] "Practical Anæsthetics for the Student and General Practitioner," by C. F. Hadfield, M.B.E., M.A., M.D.; Second Edition; 1931. London: Baillière, Tindall and Cox. Post 8vo., pp. 350. Price: 7s. 6d. net.

of the students and practitioners for whom it is written. Chapter I contains the usual short history of anæsthesia. Chapter II is entitled "General Considerations," and begins with post-operative conditions before touching on examination and preparation of the patient. There is no mention of any of the simple tests for estimating the operative risk, such as Moot's rule or the breath-holding test. Chapter III is headed "Signs and Stages of Anæsthesia—Alkaloids," each of which should have a chapter to itself. The description of the signs is clear, but undue stress is laid on the corneal reflex, that dangerous sheet anchor of the beginner. Its use is seldom required when all the other signs are properly understood. Guedel pointed out in 1920 that the third stage may be divided into substages, each of which has its own signs. Among these signs is a lateral oscillation of the globe which is characteristic of the first and second substages, and it disappears at the same time as the corneal reflex. This may be used as a substitute for the corneal reflex and the risk of trauma to the cornea be thereby eliminated. To balance these shortcomings there is an excellent short chapter devoted to "The Airway" which is a matter of supreme importance, but one that rarely has sufficient prominence. It has been truly said: "Take care of the airway and the inquests will take care of themselves."

Nitrous oxide and oxygen are discussed at length, but the use of added ether is considered almost essential for major surgical procedures. Possibly this is true of the "bubble bottle" type of apparatus which is described. Reference to this method is excused on the plea that such a machine is cheaper and the only one that the student and general practitioner are likely to use. With more accurate machines, such as the McKesson and Heidbrink, ether should seldom be required, if adequate prenarcosis be given. Ethylene receives very little space, although it has many advantages, and in some clinics has almost superseded nitrous oxide.

In the fifty pages devoted to ether the different methods of administration are carefully described. It is disappointing, however, to read that: "Perhaps the most satisfactory procedure is to commence with ether in a Clover's inhaler with the help of nitrous oxide or ethyl chloride." This seems cumbersome and unpleasant for the patient when compared with the rapid and pleasant induction with the open method by ethyl-chloride-ether sequence which is so commonly used in Australia. Also the author suggests that in the maintenance of anæsthesia by the open method ether be added in amounts of half a drachm at a time and that "the total amount used for an average operation of one hour's duration should work out at 10 or 12 ounces," whereas those who use a constant drip of ether, instead of periodic douching, usually find that six to eight ounces are sufficient for the first hour.

There is a good *résumé* on "Avertin" which contains a timely warning: "There is no doubt that 'Avertin' is a very valuable addition to the anæsthetist's armamentarium, but it will probably only remain so while it is recognized as an adjuvant to anæsthesia rather than an anæsthetic itself."

The section on local anæsthesia is short, but adequate. But the suggestion of anæsthetizing the urethra by injection of 4% to 5% solution of cocaine will not meet with general favour; this procedure has been followed by a number of deaths. The use of Ryall's solution, containing only 0·5% cocaine, is safe and provides equally good anæsthesia.

Spinal anæsthesia receives more attention than is usual in a book of this size. Dr. Hadfield shows a preference for "Stovaine," although "Novocain" is usually considered safer. "Spinocain" is reviewed by him partly on the result of Pitkin's own demonstrations in London, and quotations are made from the publications of others. Among these is the statement of Mr. Donald: "Spinocain possesses no special advantage over neocaine or novocaine in spinal analgesia." This section would be improved by more illustrations, while that of a lumbar puncture needle of the old type with a long bevelled point might be replaced by one showing the Labat or Pitkin needle with a short bevel.

Apart from these few criticisms, the book can be heartily recommended as an excellent exposition of sound and up-to-date anæsthetic teaching, eminently suitable for the student and for those practitioners who wish to keep themselves in touch with the best of modern anæsthetic work.

ANTE-NATAL SUPERVISION.

THE second edition of "Ante-Natal Care," by W. F. T. Haultain and E. Chalmers Fahmy, has now appeared.[1] It is much the same as the first, with the following additions. The Aschheim-Zondek test for pregnancy is briefly described.

There is a chapter on post-natal care; the authors almost apologize for inserting it, but it is a valuable addition and forms one of the best chapters in the book.

The other addition, "Maternity and National Health Benefits," of course, does not apply to this country.

Ante-natal care is necessary for intelligent and successful midwifery and we can recommend this book to all engaged in this branch of medicine.

DISEASES OF THE SKIN.

A SMALL work, the "Fundamentals of Dermatology," by Dr. Alfred Schalek, has proved its popularity by the appearance of the second and revised edition.[2]

In the 247 pages the author has in a concise manner given in an alphabetical arrangement the diseases of the skin. Altogether there are 58 illustrations, which are chiefly taken from photographs of typical cases.

For students and those in general practice this book should be of value. It is not a work intended for the specialist, but those who study it should read with interest the chapter on dermatological aphorisms.

PROGRESS IN UROLOGY AND DERMATOLOGY.

IN the volume on dermatology and urology of the "Practical Medicine Series," 1930, Dr. John Cunningham has contributed an excellent summary of the year's work, although suffering the necessary handicap of restricted space.[3] It will be found especially valuable to the general surgeon and practitioner who have no time to study the voluminous literature on these specialties. The extensive bibliography will be found useful to those who wish for references on every particular subject.

The part of the volume devoted to urology is arranged in six sections: general considerations, kidney, bladder, prostate, genitalia and gonorrhœa. Under "General Considerations" intravenous urography is well discussed, and the conclusions arrived at, to use the author's words, are "that a too general use of this method may take place, and is to be feared. It should be used only by those competent to interpret its portrayals and correlate them with the other procedures of urologic diagnosis." Some newer urinary antiseptics are also mentioned. The subject of renal counterbalance is dwelt on by Jielson, Back and Moritz. Their experiments do not demonstrate any renal atrophy of disuse; in fact, they strongly suggest that such does not occur. The operative treatment of prostatic obstruction is discussed, but this does not compare favourably with the rest of the volume. The same applies to the sections on the genitalia and gonorrhœa.

In the section devoted to dermatology recent work is summarized.

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[1] "Ante-Natal Care including the Abnormalities Associated with Pregnancy and a Section on Post-Natal Care," by W. F. T. Haultain and E. Chalmers Fahmy, with a foreword by R. W. Johnstone; Second Edition; 1931. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 134. Price: 5s. net.

[2] "Fundamentals of Dermatology," by Alfred Schalek, M.D.; Second Revised Edition; 1931. Philadelphia: Lea and Febiger. Demy 8vo., pp. 258, with illustrations. Price: $3.00 net.

[3] "Practical Medicine Series: Dermatology and Urology"; Series 1930. Chicago: The Year Book Publishers. Crown 8vo., pp. 474. Price: $2.25 net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 28, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

MEDICAL SERVICE AND THE COMMUNITY.

THE urgency of the hospital problem is recognized by the medical profession and the public. The general public is faced on the one hand with the need for financing hospitals, and on the other with the desire of all sections of the community to receive treatment which, they know, can best be obtained at a well equipped hospital. The medical profession knows that it is being exploited, that it is giving honorary service to people who are well able to pay for medical attendance, and that if the drift is allowed to go on, the exploitation will become worse, until a final state, not to be contemplated, is reached. It is not necessary to review the conditions which have brought about this state of affairs; to make such a review would take much space; and, in any case, most medical practitioners who read this journal, could at short notice make a fairly complete list of the contributing factors. Readers are referred again to the articles from the pen of Dr. D. M. Embelton, which have appeared from time to time in these pages. Perhaps it may be stated that one important reason for hospital bankruptcy and abuse of the honorary system, apart from any other reason that may appear inevitable or excusable, is the desire of many people to get something for nothing, a desire that is evident in every walk of life. Australia is, of course, not the only country faced with hospital problems and problems of medical service to the community. Journals from the old country have been full of reports, discussions and schemes for meeting the situation in Great Britain. So eminent an authority as Sir Robert Bolam, discussing the payment of hospital staffs, is reported as having stated that the system is "rotten" and that it is breaking down as far as the staffs are concerned. On the other hand, many contributory schemes in England are working satisfactorily, and a British Hospitals Contributory Schemes Association has recently been formed.

The medical profession adheres and will always adhere to its willingness to give gratuitous advice and treatment to the indigent. This attitude is traditional and medical practitioners would not have it otherwise. Therefore, when contributory schemes for medical services are discussed, it must be clearly understood that the indigent are excluded. The division of the Commonwealth into six States makes any attempt at discussion of the question at issue a matter of difficulty. Hospitals are controlled differently in each State and divergent views are held. Certain general principles can be stated. Medical practitioners should be paid for their services by those who can afford to pay, adequate hospital facilities should be available for those who require them, and the patient should be able to choose his medical attendant. In several States definite attempts have been made to improve matters. In Queensland a Royal Commission has recently made a report which was referred to in these columns. In this report it was stated that contributory schemes should be encouraged. In Western Australia a Hospitals Tax has been imposed. In New South Wales a proposal has been made to establish a metropolitan hospitals contribution fund. The Hospitals Commission has given details of the proposed principles of the scheme. A meeting of the New South Wales Branch of the British Medical Association has been held to consider the scheme, and in another place in this issue the resolutions passed at the meeting are published. It will be noted that the Branch is prepared to support the scheme, provided that certain conditions are observed. The basis of the scheme is a weekly

contribution, and the members of the Branch ask *inter alia* that community services be established in metropolitan hospitals. At present country practitioners in New South Wales are in a position of being able to charge patients admitted to "intermediate" and private beds, and the system is working well.

All the different schemes and methods of working at present in vogue, including the proposed New South Wales scheme, are initiated and controlled by official bodies, hospital boards or other organizations. The medical profession may be considered as a subsidiary part of the scheme; it may not be consulted; it may be called upon to cooperate and may have the opportunity of expressing its views; it will have no means of seeing that effect is given to them. The Victorian Branch of the British Medical Association has devised a scheme for the provision of hospital facilities and medical service on a contributory basis. In another place in this issue will be found a special article setting out the views of the Branch Council and giving an outline of the scheme. The arguments in favour of the proposals are unanswerable. Wisdom has been shown in dividing the proposed organization into two parts and investing the control of the funds to be used for nursing and hospital service and consulting and specialist service in representatives of non-medical bodies, as well as in representatives of the medical and nursing professions. By this scheme, hospitals, medical attendants and pharmacists will be paid for their services. There will be no abuse unless control is lax. The scheme is one of approved insurance. Moreover, it is insurance at the instance of the medical profession, and it gives each insured person the status of a private patient. These considerations make the scheme most desirable. The success of any hospital, its reputation as a "great" institution or its mediocrity, depends entirely on the standard set by the medical staff. So will it be with the working of this scheme. Medical practitioners of all States will wish to see it carried to success. The members of the Victorian Branch have the matter in their own hands. If they succeed, they will establish a new era in medical practice in Australia.

Current Comment.

THE NEUTROPHILE LEUCOCYTES IN ACUTE INFECTIOUS DISEASE.

IN 1904 Arneth, investigating the nuclear structure of neutrophile cells in infection, observed that there seemed to be a constant relation between the intensity of the infection and the number of non-segmented neutrophile cells in the blood. In very severe infections neutrophile cells with nuclei showing three, four or five lobes would disappear, being replaced by cells whose nuclei were non-segmented. With decline of the infection mature neutrophile cells rapidly replaced those with non-segmented nuclei. V. Schilling did not employ subclasses of neutrophile cells which depended on the number and shape of nuclear segments, but included all the segmented forms into one group—the "segmented." The non-segmented were divided into two subclasses. One is the *"Stabkernige"* subclass of immature neutrophile cells, the nuclei of which are unsegmented, but are thin and assume many forms. The other subclass (*"Jungkernige"*) has nuclei usually sausage-shaped, with no evidence of approaching segmentation. Arthur Weiss[1] records the examination of about 20,000 blood smears taken from patients with a variety of medical and surgical infections. He gives full details of cases of lobar pneumonia, appendicitis with peritonitis and bronchopneumonia, subhepatic abscess, perisigmoid abscess, gall-stones with ruptured gall-bladder. Weiss observes that an older teaching was that all infections resulted in leucocytosis, with increase of polymorphonuclear cells. It was held that the larger the number of leucocytes the better was the patient's resistance against the invading organism, and the higher the percentage of neutrophile cells the severer was the type of infection. If the leucocyte count was relatively small but the proportion of neutrophile cells high, it was thought that the patient's resistance was overcome by the infection and the issue was usually fatal. The other leucocytic constituents were deemed to play merely a passive part and any variation in their number was thought to depend on a concomitant variation in the neutrophile cells. The observations of Weiss, however, indicate that the neutrophile cells are not the sole active participants. Instead of the invading organism exerting (by means of its toxins) a chemotactic force giving rise to neutrophilia, there is a definite sequence of coordinated biological events, each unit having its definite time and place. As each cellular system is required to act, the others recede into the background, to reappear when necessary. The bone marrow, the reticulo-endothelial system and the lymphatic system all play their respective parts. With the onset of an acute infection the neutrophile cells are first called into activity. The bone marrow, which has been manufacturing segmented neutrophile and non-segmented or "staff" cells, is suddenly required to supply more

[1] *Archives of Internal Medicine*, September, 1931.

cells. When the demand is mild, the bone marrow is able to increase its supply and there is an augmentation of leucocytes in the form of mature neutrophile cells. The non-segmented ("staff") neutrophile cells may or may not be slightly increased. But if the call on the bone marrow be too great for its normal productive forces, there will be a moderate or great increase in the staff forms, depending on the severity of the infection. At the crisis of pneumonia the staff cells may total 70% or more of the whole leucocytes. While this neutrophile phase is predominant all the eosinophile cells will have left the circulation; the monocytes also will have gone or will remain only in small numbers. Similarly the lymphocytes conspicuously diminish. After surgical removal of the infective focus or expulsion of the causative organisms the neutrophile phase passes, to be replaced by monocytes. These cells were formerly believed to be of myeloid origin, but many observers consider that they represent a definite cellular system of hæmatopoiesis. Monocytes generally are poured into the blood when the peak of the staff cells is just beginning to decline. In subacute and chronic cases monocytes will be increased until convaleseence. The monocytic phase, being of short duration, may be missed unless frequent blood examinations are made during the crisis. The lymphatic system is the last to come into play. Normally, the lymphocytes constitute 25% to 30%. During the height of infection they drop to between 1% and 10%. During convalescence they gradually rise and may total 50% to 60%. The increase in the different cellular elements at different phases of an infection is not passive. There is evidence of marked activity of the monocytes, which may be observed with vacuoles, phagocytosed remnants and mitoses. Lymphocytes also display hyperactivity by the presence of Rieder forms, Türck irritation cells and double nuclei.

Weiss concludes that leucocytosis caused by acute infections is primarily the result of stimulation of the bone marrow. The reaction of the bone marrow is non-specific and depends on the type of organism and the degree of irritation caused by bacterial toxins. The peak of the staff count and the height of infection usually coincide. The peak of the staff count drops when the infectious process is removed or overcome. A persisting high staff count generally indicates a complication, or it may mean that the infection is becoming chronic or subacute. Further, a persisting high staff count, without the possibility of removing the infective process, generally presages a fatal issue. A fatal issue is also usually indicated by a high staff count early in the course of lobar pneumonia. More accurate information is furnished of the course of an infection by the staff count than by the temperature chart. The staff count is more reliable than the leucocytic or polymorphonuclear count. The eosinophile cells return into the circulation coincidentally with the sharp drop in the staff count and sudden increase in the monocytes. If the infection becomes subacute or chronic, there may be a persistently elevated staff count together with an increased number of monocytes and lymphocytes. During the neutrophilic stage of acute infections there is lymphopenia, which is replaced by lymphocytosis during convalescence and healing. In subacute or chronic infections lymphocytes are generally increased. One single report of a high staff count does not necessarily indicate a fatal issue. Daily cell examinations should be made, as prognosis depends on the curve, and blood changes must be considered with the clinical picture. But conspicuous blood changes cannot be disregarded in the absence of clinical confirmation. Also a definite clinical picture cannot be negated because of a lack of confirmatory hæmatological findings. Occasionally, for no obvious reason, the blood picture does not indicate the patient's condition.

The observations of Weiss are of great interest and importance, and further research on the same lines is required. This might be profitably extended to such disorders as typhoid fever, pertussis, mumps, syphilis and others. In mumps we know that the leucocyte count varies very greatly. It has been described as normal or low or high, particularly when complicated by orchitis. In this disorder also there may be a lymphocytosis or increase of polymorphonuclear leucocytes. In typhoid fever there is a leucopenia with a relative lymphocytosis, lasting till convalescence. The number of polymorphonuclear cells is usually lowered. When complications exist, such as hæmorrhage, peritonitis, parotitis or otitis, there is often a leucocytosis and increase in polymorphonuclear cells. In pertussis there is an initial and terminal leucopenia; but when the paroxysmal stage is at its height there is a characteristic leucocytosis and lymphocytosis. In all such diseases the staff cells might be estimated as regards their bearing on the course and termination of the disease. So, too, in syphilis, similar investigations might be made and their relation noted to clinical improvement or alteration in the serological reactions.

CICATRICIAL STENOSIS OF THE ŒSOPHAGUS.

GEORGES PORTMANN has reported an "autodilatation" method of treatment of cicatricial stenosis of the œsophagus.[1] This method is ingenious and apparently safe. Dilatation is practised "by the patient himself," the progression of the dilator taking place merely under the peristaltic movements of the œsophageal walls. Gastrostomy is performed. A filiform bougie is passed at a later stage into the stomach with the aid of the œsophagoscope. The end of the bougie is picked up in the stomach by a cystoscope inserted through the gastrostomy opening. A silken thread is fastened to the bougie, which is withdrawn from the mouth and established as an endless thread. Dilatory metal olives fixed on the endless thread take three to six days to pass the stricture. Treatment extends over months or years, and Portmann has treated seven patients successfully.

[1] *Proceedings of the Royal Society of Medicine*, September, 1931.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Bacterial or Infective Endocarditis.

WILLIAM S. THAYER (*Edinburgh Medical Journal*, April and May, 1931) discusses bacterial or infective endocarditis. He bases his observations on 306 cases of ætiologically proved bacterial endocarditis occurring in the Johns Hopkins Hospital during the last forty years and on a few cases met with in private practice. He states that whereas rheumatic endocarditis arises through the development of minute foci of necrosis in the substance of valve or heart wall accompanied by an inflammatory reaction which spreads to subjacent endocardium, bacterial endocarditis presents its earliest changes on the surface of the endocardium. By far the commonest ætiological agent in the latter condition is the streptococcus, which is found in over 60% of cases and was present in 109 of the author's series. In the acute type the β hæmolytic streptococci usually occur; in the subacute forms *Streptococcus viridans* or α hæmolytic streptococci are found, and of these *Streptococcus salivarius* is the commonest. The left side of the heart is attacked most frequently in streptococcal and rheumatic endocarditis in contrast to the other forms of bacterial endocarditis; the mitral valve is the seat of election in streptococcal infections. In acute endocarditis the course is that of an intense septicæmia following puerperal sepsis, infected wounds, empyemata *et cetera*. In the subacute infections the portal of entry was apparent only in 27% of cases, but in many of these, teeth and tonsils and sinuses were under suspicion. The author points out that transient bacteriæmia in infections with the milder forms of streptococci is not uncommon, and the recovery of such an organism from the blood, even on several occasions, does not justify a diagnosis of subacute vegetative endocarditis. He also stresses the necessity for keeping media inoculated from blood under observation for at least three weeks before giving a negative finding. Next to the streptococcus the pneumococcus is generally recognized as the most frequent ætiological factor. It was present in thirty-eight of the author's cases. The course of infection is as a rule characteristically acute and rapidly fatal, with extensive destruction of the valves. The portal of entry was most commonly an acute pneumonia or bronchopneumonia; seldom is the source of infection unrecognized. In gonococcal infections, of which there were thirty-one cases in the series, an acute destructive process usually attacks previously unaffected valves. There is profound intoxication with anæmia and high leucocytosis. Recovery may occur, but is unusual. The author presents the view that in individuals between the ages of eighteen and thirty, with characteristic manifestations of endocarditis and repeated failure to recover organisms by culture from the blood, the possibility of a gonococcal infection should be considered. After streptococci and pneumococci, *Staphylococcus aureus* is the most common ætiological factor. This organism was present in twenty-eight of the author's cases. It is preeminently a terminal invader, accompanying a general sepsis associated with a focal infection, such as osteomyelitis, carbuncle or puerperal sepsis. Suppurative metastases are common, and the tricuspid valve is relatively frequently attacked. Infection by this organism is almost invariably fatal, and runs a rapid course. *Staphylococcus albus* is an occasional causal agent, but the author points out the necessity for careful bacteriological technique before this organism is accepted as causal. *Bacillus influenzæ* occurred in nine cases of the series, while *Bacillus pyocyaneus*, *Bacillus mucosus capsulatis* and *Bacillus anthracis* were each recovered once. In the matter of treatment the author suggests that specific sera should be tried in streptococcal, pneumococcal and meningococcal infections, while in gonococcal infections transfusions with blood from one recently recovered from a febrile complication of gonorrhœa should be used. He deprecates the intravenous use of internal antiseptics, and on Capp's recommendation gives cacodylate of sodium subcutaneously.

Isolation of Brucella Abortus from a Human Fœtus.

C. M. CARPENTER AND R. BOAK (*The Journal of the American Medical Association*, April 11, 1931) in 1927 isolated *Brucella abortus* from a human fœtus. Bacteriological examinations of twenty-eight fœtuses and thirty-four placentæ were made. In fourteen instances the placenta and fœtus came from the same patient. The ages of the fœtuses varied from thirty days to seven months. Incubation of material from eleven fœtuses and seven placentæ yielded no organisms. Various organisms were recovered from the remainder. In only one was the *Brucella abortus* found. A full history of the patient is given. The *Brucella abortus* was identified by culture, antigen and agglutinin tests and guinea-pig inoculation with repetition of the tests on the organism recovered. The close biological relationship between *Brucella abortus* and *Brucella melitensis* renders it impossible to describe the pathology of the two organisms separately. Numerous abstracts from and references to the literature of the subject are given. This is the first instance in which the *Brucella abortus* has been isolated from the human fœtus. The authors were not able to determine whether the placenta was also affected or to study it histologically, but they believe that it must have been infected and that, although the data are incomplete, the abortion was due to the *Brucella abortus*. The patient did not give a history of undulant fever, her blood was not examined for the organism, and they did not have an opportunity of examining her after she left hospital. Reports of the isolation of *Brucella abortus* from the genital tracts of men and women indicate that it is not a rare cause of disease of the reproductive organs. This should be borne in mind by those investigating the pathology of the genito-urinary system.

Induction of Lymphocytosis and Lymphatic Hyperplasia.

BRUCE K. WISEMAN (*Journal of Experimental Medicine*, April, 1931) records observations on the lymphocytosis and lymphatic hyperplasia produced by injecting rabbits with protein. The proteins used were extract of chick embryo, egg albumen, and normal horse serum. The injections were made intraperitoneally, intravenously and subcutaneously. Injections were made for the most part every day for six days each week. All the forms of protein used elicited hyperplasia of the spleen, accompanied by a lymphocytosis in the peripheral blood. The greatest response seemed to be obtained by the intravenous route, and egg albumen appeared to be the most potent of the substances used. The first increase of lymphocytes became evident between the seventh and tenth days after injections were begun. That the fluctuations in the lymphocyte increase were independent of the spleen was shown by a study of an animal from which the spleen was removed and in which similar phenomena were noted. The increase in lymphocytes varied from 23% to 139%. At autopsy lymph glands and spleens showed hyperplasia, but the thymus appeared normal. The author thinks it probable that there is a more definitely intimate relation between certain phases of protein reaction or intoxication in the body and the lymphatic tissue than has been recognized.

HYGIENE.

The Prevention of Venereal Disease.

ARTHUR MASSEY (*Journal of State Medicine*, February, 1931) outlines some of the difficulties to be faced in practical attempts to control venereal disease. Whereas instinct usually runs parallel with inclination, inclination usually is opposed to convention, which, in the form of self-discipline, has to be regarded as the first means of attack on venereal disease. Recently public attitude towards convention has altered and irregular sex relationships are regarded more lightly. Scarcity of employment and rise in the cost of living have resulted in an increase in the average age at marriage. The ease with which contraceptives and preventive devices can be obtained has dissipated the fear of consequences which hitherto aided in lessening venereal

disease. Preventive measures may be divided into (*a*) social, (*b*) medical, and (*c*) legislative. In connexion with the first, outdoor games and exercise help to dissipate the sex instinct in the young, work against alcoholic excesses assists in adults to prevent venereal disease. Educational measures instructing parents and children are of value. Medical measures need little comment. It is hoped that public opinion will permit soon the legal requirement of a medical certificate of fitness prior to marriage. The notification of venereal disease has proved invaluable in Australian States. The incidence of syphilis has decreased noticeably there in the last ten years. Legislation to compel treatment of venereal disease appears to be useful also.

The Reduction of Mine Air Temperatures.

T. BEDFORD AND C. G. WARNER (*Journal of Industrial Hygiene*, April, 1931) describe an experiment in local air conditioning in coal mines by means of artificial humidification. The increasing depth of mining operations has caused a great deal of reseaerch as to the best means for reducing the air temperature. The humidifying apparatus was used in two positions on a coal face of 170 yards: first, 120 yards from the face and, secondly, close to the face. The better effect was obtained in the near position, but the results were not sufficiently good to warrant general application of the system.

Asbestos Dust and Primary Tuberculous Infection.

L. U. GARDNER AND D. E. CUMMINGS (*Journal of Industrial Hygiene*, February, 1931) have used fine asbestos dust in inhalation experiments on guinea-pigs, white rats and rabbits. One group of animals was infected with tubercle bacilli four days prior to the commencement of dust inhalation, while twenty-three inoculated animals were kept free from dust as controls. Twelve dusted and twelve normal animals were inoculated at a later stage and kept free from further dust. Animals from these groups have been examined by X rays, killed and examined pathologically at intervals during the experiment up to 880 days. Of the first group 26% died of epizootic pneumonia, and a similar number died of other conditions, such as enteritis and peritonitis. Pulmonary fibrosis of a nature sufficiently coarse to be visible, appeared after one year of dusting. After more than two years subpleural nodules, uniformly distributed, were visible over the surface of the lung, and in its substance similar nodules were observed on section, with pigmentation of the tracheobronchial lymph glands. Radiographic change was first noted after two years as a very fine mottling.

L. U. GARDNER AND D. E. CUMMINGS (*Journal of Industrial Hygiene*, March, 1931) report the results of a series of experiments in which guinea-pigs have been exposed to inhalation of an atmosphere containing asbestos particles for eight hours daily for as long as two and one-third years. The experiments demonstrate that fibrous structures as long as 200 microns can pass the protective mechanism of the upper respiratory tract and enter the lung without apparent evidence of injury to this mechanism. The asbestos dust does not penetrate the alveoli, but is held in the major bronchioles, and phagocytosis takes place there. Phagocytes containing asbestos particles can migrate to the lateral alveoli. In the guinea-pig fibrosis of the walls of the respiratory bronchioles and the lateral alveoli is seen after five hundred days' exposure and thereafter progressively increases in intensity and extent. Asbestos bodies are found after seventy days' exposure in the lungs. These bodies are not present in dust previous to inhalation or injection. Their formation is the first direct evidence that the body is capable of effecting changes in inhaled silica particles. Lymph stasis plays little part in early asbestosis, the structure of the dust tending to localize it within the lung. Primary tuberculous infection is influenced only to a limited degree by inhaled asbestos. At autopsy 40% of a group of primarily infected animals showed healed fibrous tuberculosis with macroscopical disease in the spleen and liver in some cases. The outcome of inoculating a group of animals with tuberculosis after inhalation has not yet been observed. The combined action of asbestos dust and tubercle bacilli in the lung produced more fibrosis than did either agent acting independently.

The Behaviour of Tetra-Ethyl Lead in the Animal Organism.

R. A. KEHOE AND F. THAMANN (*American Journal of Hygiene*, March, 1931) have investigated the rate of absorption of tetra-ethyl lead through the skin under varying conditions, its fate in the tissues and the rate of excretion. Chemically pure tetra-ethyl lead was applied to the skin of the belly of rabbits after the hair had been clipped. The skin was not shaved and no water was applied. Precautions were adopted to prevent inhalation of the lead by the animal. The skin was cleansed after a definite period by sponging with kerosene. In a further series of experiments with ethylized petrol the rabbit's foot was immersed in a glass tube containing the solution. The experiments proved the absorption of tetra-ethyl lead through the skin, and its distribution in the tissues initially in a manner similar to that occurring with an oil-soluble material. In the tissues, however, it is rapidly decomposed, and after three to fourteen days is distributed in a manner characteristic of that occurring with a water-soluble compound. When small doses are used, the rapid decomposition prevents its primary absorption by the nervous system. The excretion follows quantitatively that of water-soluble lead compounds, indicating that poisoning by tetra-ethyl lead is not different from poisoning by other lead compounds. Absorption of tetra-ethyl lead from petrol in concentrations not in excess of 0·1% is inappreciable.

Prevention of Industrial Disease.

E. L. COLLIS (*Journal of State Medicine*, May, 1931) points out that the prevention of disease in industry may be more closely concerned with an undue prevalence of sickness of usual types among industrial workers than with risks or hazards peculiar to industry. The grouping of population in industry affords exceptional opportunities for the study of the effects of various influences on health. The personal and the environmental factors must be considered to a large extent separately, although they may interact. Certain diseases, such as rheumatism, are accentuated by occupational environment, while others are so specific that they hardly exist apart from the occupation. In recent years the scope of industrial medicine has widened and active means and standards for the prevention of sickness have been formulated. It is realized that whatsoever interferes with health, interferes with output. The labour turnover, or labour wastage, caused by drifting of employees from one industry to another is bad for the workers and their work. Much time is lost from sickness and accidents by employees who are becoming accustomed to an industry. It is consequently necessary to reduce labour turnover to a minimum in order to reduce sickness. Excessive heat, cold, or humidity, lack of facilities for cleanliness, bad or insufficient lighting, or exposure to excess of heat or light rays from molten metals, long hours of work, or incorrect distribution of spells are some of the many factors influencing industrial health. The importance of dust, noxious fumes and gases as agents causing disease must not be overlooked. Age and sex are important influences affecting sickness.

The International Factory Clinic.

A. ROLLIER (*Journal of State Medicine*, April, 1931) describes the International Factory Clinic at Leysin, Switzerland, which was opened in February, 1930, to help necessitous patients suffering from surgical tuberculosis. In this clinic heliotherapy is combined with manual occupation to effect a cure. The effect of the sun treatment on the mental and moral outlook of the patients is striking, and the influence of occupation has proved a valuable therapeutic factor. Intellectual study is combined with easy manual work in carefully graded doses. Baskets, light furniture *et cetera* are manufactured by the patients, who receive the sale price of the articles less 10% to cover costs. The factory is built on hygienic lines with "Vita" glass windows. Patients of all nationalities are admitted. The results have been found most encouraging.

Special Articles on Aids to Diagnosis.

(Contributed by Request.)

VIII.

THE SCHICK TEST.

THE function of the Schick test is to disclose the fact of the susceptibility or otherwise of an individual to the diphtheria toxin and therefore his susceptibility to the disease.

Technique of the Schick Test.

The technique of the test is so well known as scarcely to bear repetition. The toxin is issued by the Commonwealth Serum Laboratories in small phials. The phial is sterilized by placing it in lysol or spirit. After being thoroughly washed in sterile saline solution, it is placed in a sterile bottle, the amount of 0·85% saline solution recommended in the instructions issued with the product is added, and the phial is broken thoroughly with a glass rod or forceps. Of the resultant mixture 0·2 cubic centimetre contains one-fiftieth of a minimal lethal dose of the toxin and is the amount required for the test. For purposes of control, one half of this quantity may be heated to 70° C. for five minutes, the toxin thus being destroyed and the protein constituents being left unharmed. With the present day use of anatoxin immunization this is usually omitted, the anatoxin skin sensitivity test taking its place. The skin over the forearm is cleansed with methylated spirits, and the needle of the hypodermic syringe "wriggled" into the skin with the bevel facing upwards. It is important that the syringe should not leak back, as this may result in insufficient dosage.

An accurate injection is shown by the presence of a small white wheal, its surface being pitted where it is anchored down by hair follicles and sweat ducts. No dressing is required.

Types of Reaction.

The types of response observed are: (i) No reaction, (ii) positive reaction, (iii) pseudo (no reaction and pseudo), (iv) positive and pseudo.

1. In this group nothing is observed on either arm at any time.

2. When the positive reaction occurs, nothing is observed on the control arm at any time. On the test arm, after twenty-four to thirty-six hours, a circumscribed flush develops, reaching its maximum in four days. Slight swelling and infiltration are present, and the redness slowly fades to a brownish tint, leaving an area of desquamation. The pigmentation may last for weeks.

3. The pseudo-reaction appears in twenty-four hours, which is earlier than the Schick reaction. It is shown as a red, rather diffuse, inflammatory area, equal in extent on both arms, and is less circumscribed and more "angry looking" in appearance than the true reaction to the Schick test. By the fourth day it has disappeared.

4. The positive and pseudo-reaction is more difficult to interpret. The pseudo-reaction develops in twenty-four hours, a deeper coloured, more circumscribed red area appearing in the centre. The outlying inflammation subsides by the fourth day, leaving a typical reaction. On the control arm the redness has disappeared. If only one reading can be taken, this should be done on the fourth day, but fortunately this type of reaction is rare, and occurs mainly amongst adults.

Pseudo-reactions are met with much more commonly among the "immune" population and the theory held at present is that the reaction is a true allergic response to the protein of the diphtheria bacillus, and indicates a certain degree of immunity.

Practical Values of the Schick Test.

The Schick test is used as a preliminary to mass immunization of children of school age. When toxin and antitoxin immunization was the method mainly used, the Schick test was omitted in young children, as the great bulk of them were reactors, and the omission of the test saved time and expense. With the present day use of anatoxin immunization, as it is essential to perform a skin sensitivity test before proceeding further, it is considered advisable to carry out the Schick test at the same time, the sensitivity test acting as a control.

The test is used after a suitable interval to discover whether immunization has been effective. All previous positive reactors should fail to react some months after artificial immunization.

All nurses entering hospitals for training should be tested by the Schick test upon arrival and the reactors immunized.

Schick testing combined with swabbing is used in the following circumstances. On the occurrence of an outbreak of diphtheria in an institution it is advisable to combine the Schick test with swabbing of throats and noses. All organisms morphologically resembling the diphtheria bacillus grown from swabs should be tested for virulence. Those individuals who fail to react to the Schick test and who grow a diphtheria bacillus from nose or throat are dangerous and must be immediately isolated pending the performance of a virulence test. If the organism is proved virulent, they must be kept isolated and treated until normal swabs are obtained. On the other hand, Schick positive reactors with virulent diphtheria bacilli in throat or nose either have the disease or are incubating it, and should be treated accordingly.

Artificial Immunization.

Artificial immunization is of two kinds, passive and active.

Passive Immunization.

By injecting subcutaneously 1,000 units of antitoxin it is possible to immunize an individual passively for about a period of three weeks. This method is of use when it is desired to protect for a short time delicate children or those suffering from illness and whom it is undesirable to immunize actively. It may also be used for a baby who is being breast fed by a mother suffering from clinical diphtheria.

Active Immunization.

By the injection of small quantities of a suitable antigen an individual can be made to produce his own antibody. In the case of diphtheria the toxin is the natural antigen, but in practice this is of necessity dangerous and other methods have been devised. Of these the most widely used are a mixture of toxin and antitoxin in which the toxin is slightly over-neutralized by antitoxin or some form of detoxicated toxin or toxoid; anatoxin or formalinized toxin is an example of this.

Toxin-Antitoxin Mixture.—In the product issued by the Commonwealth Serum Laboratories each cubic centimetre of the mixture contains one unit of antitoxin and 0·8 of a lethal dose of diphtheria toxin. This means that there is 25% more antitoxin present than is required to neutralize the toxin. Injections of one-eighth, one-quarter and then one-half of a cubic centimetre are given subcutaneously at intervals of five to seven days. Individuals giving a plain positive Schick reaction show no ill effects from these injections, but the positive and pseudo reactors may develop local tenderness and swelling owing to their sensitivity to the bacillary protein as indicated by the pseudo-reaction. Individuals retested after an interval of six months should fail to react to the Schick test.

Anatoxin Immunization.—Anatoxin is produced by the action of formalin upon the diphtheria toxin. The toxicity is destroyed by this action, but the antigenic value is retained. It is therefore considered a safer and more efficacious method of producing immunity.

Certain members of the community, mainly older children and adults, show a sensitivity to anatoxin. Therefore, it is desirable to perform an anatoxin skin sensitivity test before proceeding with immunization. It is convenient in order to save delay to carry out the Schick test and the anatoxin sensitivity test at the same time. A one in 20 dilution of anatoxin for the test is supplied by the

Commonwealth Serum Laboratories and a comprehensive booklet of instructions is issued with the product. An intradermal injection of 0·1 cubic centimetre of the diluted anatoxin is given into the forearm. A positive reaction is shown by an area of redness of more than 1·25 centimetres (half an inch), which reaches its maximum in twenty-four hours and should be read then. The Schick test is observed at the same time, but must be read three days later. The types of reaction are four in number:

1. Anatoxin no reaction; Schick no reaction.
2. Anatoxin positive; Schick no reaction.
3. Anatoxin no reaction; Schick positive.
4. Anatoxin positive; Schick positive.

The first two groups may be disregarded, as immunization is unnecessary.

In the third group immunization with anatoxin should be proceeded with as soon as possible. The Commonwealth Serum Laboratories advise two subcutaneous injections, the first of 0·5 cubic centimetre and the second, three weeks later, of one cubic centimetre of the product. If it is desired to give a third dose, this should be one cubic centimetre given three weeks after the second dose. Immunity is produced in a large percentage of cases.

In the fourth group, in which the patient reacts to the Schick toxin and is also sensitive to anatoxin, it is necessary to move warily. The anatoxin skin sensitivity test, as already explained, is considered to be a true allergic response on the part of the individual to the protein in the bacillary bodies, and indicates that he has already encountered the organism, possibly through a mild, unrecognized attack of clinical diphtheria. Reactors are therefore believed to have a certain degree of immunity. Fortunately this type of case is rare, and as unfavourable results have been reported as following on anatoxin injections in these individuals, it is not considered advisable to proceed with immunization.

Immunity in a population is relative and not absolute. Those members who fail to react to the Schick test may be considered to have a sufficiency of circulating antitoxin in the blood or, what is perhaps more important, as having their cells well trained, ready immediately and freely to pour out antitoxin in response to an antigenic stimulus, such as the toxin of the Schick test. On the other hand, those individuals who react definitely to the Schick toxin, are devoid of circulating antitoxin and unable to manufacture it.

Between these extremes is a large mass of the population whose immunity depends on many factors, important among which are the massiveness of the infection and the degree of antitoxin forming power in the attacked cells.

It is among this group that the fallacies of artificial immunization occur.

HELEN KELSEY, M.B., B.Sc., D.P.H. (Melb.),
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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on September 24, 1931, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. GEORGE BELL, the President, in the chair.

Encephalomyelitis.

DR. A. B. K. WATKINS read a paper entitled: "Surgical Considerations in Inflammatory Diseases of the Brain and Its Coverings" (see page 667).

DR. PHYLLIS M. ANDERSON read a paper entitled: "A Modern Conception of Meningeal Inflammation" (see page 672).

DR. OLIVER LATHAM read a paper entitled: "Some Thoughts on the Term Acute Disseminated Encephalomyelitis and Its Affinities" (see page 677).

PROFESSOR W. S. DAWSON expressed his appreciation of the lucid and interesting way in which the three papers had been presented. They were all of such a highly technical character that the mere clinician would be chary of offering criticism. It was curious that pathologists seemed to take a morbid delight in their findings. Professor Dawson remembered a distinguished pathologist in London dancing about the laboratory with joy because he had discovered a pneumococcus in the cerebro-spinal fluid.

In regard to Dr. Anderson's paper, referring to infections of the brain, one matter of interest lay in the work that had been done on the possible pathological basis of some confusional states. It had been shown that infection might pass from chronically infected sinuses to the base of the brain. Professor Dawson referred to observations of Yates, of Edinburgh, who noticed that there was a paralysis of cilia in the upper part of the nasal mucosa in encephalitis. Le Gros Clark had made an attempt to trace the path of infection through the cribriform plate by injection into the nasal cavities of a solution of potassium ferrocyanide and iron ammonium citrate. Dr. Latham and Professor Dawson had tried to repeat the experiment and were able to notice particles of Prussian blue actually in the substance of the olfactory bulb. When they tried to inject cultures of staphylococci into the nasal cavity, they were unsuccessful in demonstrating passage of the organisms. Professor Dawson thought that possibly a selective action was involved.

Dr. Latham in his paper had shown the complex nature of brain changes. Professor Dawson stressed the possibilities that lay in this work. It had only recently been realized that the brain reacted in its own particular way to different types of inflammation. Spielmeyer had shown before Kinnier Wilson that in the brain there might be diverse reactions to the same types of noxious agents. For instance, in pyogenic infection there was often a pure glial reaction without vascular change.

Professor Dawson asked Dr. Watkins why he was averse to performing repeated lumbar puncture in meningitis and whether he had had any experience of lavage of the spinal cord through cisternal puncture and lumbar puncture. Why was lumbar puncture contraindicated?

DR. A. E. MILLS expressed the opinion that Dr. Anderson's paper was a model of lucidity and indeed of everything that was good; it was a long time since he had heard anything so clear and logical. One important point, however, had been omitted. In regard to increased intracranial pressure and outpouring of cerebro-spinal fluid, Dr. Mills was rather disappointed that no one had made allusion to the outpouring of cerebro-spinal fluid in cases of *encephalitis lethargica*. He mentioned the case of a little girl who had the condition of increased intracranial pressure. She was very young. When the condition was first noticed it was possible to take away cerebro-spinal fluid by lumbar puncture every week or ten days. Meantime they were unable to prevent increase of intracranial pressure and the patient developed convulsions. Lumbar puncture was stopped. Chronic hydrocephalus appeared. The child, however, lived; it would have been better if she had died. This condition was not tuberculous. Why was there such an outpouring of fluid in encephalitis? Why did the chorioid plexus keep on secreting? This question had arisen at the congress in Melbourne. Why was there no explanation? The suggestion might be put forward that in some cases of *encephalitis lethargica* organisms attacked and altered the chorioid plexus, which bore the brunt of the attack and which was so altered that there occurred an outpouring of fluid. But this was not a complete explanation, since outpourings might occur in other parts of the body. It was most extraordinary. Perhaps Dr. Latham could offer some explanation.

DR. A. H. TEBBUTT said that the diseases under discussion were amongst the failures of medicine and surgery. In reply to Professor Dawson he would say that the pathologist did not jump for joy at finding a pneumococcus in the cerebro-spinal fluid for any other reason than that he was able thereby to confirm the physician's diagnosis of meningitis. Dr. Anderson did not mention one point

in the circulation of the cerebro-spinal fluid which was of importance, namely, the direction of flow in the perivascular channels. These channels surrounding the vessels entering the brain were continuous with the subarachnoid space and appeared to end in the Virchow-Robin perineuronal space. Mott contended that the flow was from the subarachnoid space inwards, but the work of Weed and others had shown that the flow was in the reverse direction. It seemed to him that in cases of meningitis, for example, where there was marked increase of cerebro-spinal fluid under considerable pressure, unrelieved by lumbar puncture, organisms and pus cells might be forced up these channels and increase the encephalitis. It had long been held that the intrathecal route of administration of convalescent serum in poliomyelitis was the most effective. One wondered how this serum in the subarachnoid space could reach the interior of the spinal cord. One could scarcely imagine it passing directly through the pia, and the direction of flow in the perivascular channels would not assist its reaching the interior of the cord. One would think that it would be better to give the serum intravenously in large doses. It would then circulate through all the vessels in the central nervous system. However, the intrathecal route was still being used.

Dr. Tebbutt said that he might have misunderstood Dr. Anderson, but he gathered that she held that the leucocytes in the subarachnoid space in meningitis came through the chorioid plexus. It was known, however, that in localized serous meningitis there might be numerous leucocytes in the cerebro-spinal fluid, and these undoubtedly came from the inflamed membranes. It seemed reasonable, therefore, that the majority of leucocytes in generalized meningitis came from the membranes of the brain. He agreed with Dr. Anderson that influenzal meningitis was a very fatal disease. Meningococcal meningitis was in his experience rare in Sydney. In a recent publication by the Ministry of Health in Great Britain it was shown that there were from three hundred to five hundred deaths each year from this type of meningitis, and there were some reports that the serum treatment was unsatisfactory. It was thought that there might be considerable variation in the races of meningococci now causing infection, as compared with those worked out in the epidemics during the war period. There were no type sera available in Australia. In treating the few sporadic cases that now occurred, one had some doubt as to the specificity of the sera obtainable. If an epidemic occurred here, it was possible that they might obtain disappointing results. A number of cases of mild infections, apparently, with streptococci and pneumococci probably not blood-borne, had been reported in which recovery had taken place, so that the prognosis was not always hopeless in these infections.

DR. GARNET HALLORAN thanked the readers of the papers. He particularly thanked Dr. Watkins for giving prominence to the question of drainage of the cisternæ in acute suppurative meningitis of otitic origin. Dr. Watkins had mentioned a paper by Stewart in which were cited 114 cases of acute purulent meningitis of aural origin treated at the Edinburgh Infirmary, mostly by Logan Turner and Fraser. Dr. Halloran felt that he would like to differ in the interpretation of this paper. Evidently Dr. Watkins had concluded that the number of recoveries when there were demonstrable organisms in the cerebro-spinal fluid was so small that operation was contraindicated. Dr. Halloran had interpreted this the other way about; he thought that the patient had a reasonable chance of recovery if operation were undertaken in such conditions. Among the 114 cases spread over twenty years, eight patients recovered, all after operation. Four had organisms in the cerebro-spinal fluid, from three of which organisms were grown on culture. The organisms, if any, were not named in the remaining four, but they were included in this group of cases of acute purulent meningitis.

The lesions found at mastoid operation were gross and had included brain abscesses. It was obvious that these patients could not possibly have recovered unless operation by the mastoid route had been undertaken. In no case were the cisternæ stated to have been opened. Also the conditions of all these patients, except one who recovered, were due to chronic suppurative *otitis media*. Only one was secondary to acute *otitis media*.

Dr. Halloran could add two more recoveries, both from chronic *otitis media* with organisms on lumbar puncture, which had occurred in Sydney. In one of these the labyrinth was drained at that time. Out of the 116 patients it was seen that ten recovered, six having had demonstrable organisms on lumbar puncture. Dr. Halloran felt that it was better to give these patients a chance; six in 116 recovering after operation by the mastoid route. If he had such a lesion himself, he would hope to be given this chance. Therefore, operation should be undertaken in cases of chronic suppurative *otitis media* with suppurative meningitis to the extent at least of exposing the meninges.

It was stated that of the 114 meningeal infections, 31% were from acute *otitis media*. In this latter type, operation was hopeless. Eliminating these, the percentage of cures following chronic *otitis media* would be higher. Another point was the hopelessness of operative procedure in sphenoidal meningitis. In this respect it was possibly analogous to osteomyelitis of the frontal bone, though osteomyelitis of the frontal bone was not absolutely hopeless. It was also analogous to osteomyelitis of the petrous tip of the temporal bone with Gradenigro's syndrome, in which there was a reasonable chance of recovery. Obviously, in cases where the lesion was inaccessible to surgical procedures, as when the diploe of the sphenoid were invaded, it was hopeless to indulge in operation.

Dr. Watkins, in reply to Professor Dawson's question as to why he stressed the point that repeated lumbar puncture was bad in meningitis, said that it was not so much repeated as large or massive lumbar puncture that was to be deprecated. Most of the patients with meningitis who recovered, had a local lesion, and it was important that it should be treated before the infection became generalized. If the involvement was limited above, it might be possible to keep it so. By tapping the cerebro-spinal fluid below, in the lumbar region, the fluid was caused to flow in a downward direction, thus reversing the normal flow of the cerebro-spinal fluid and bringing the organisms down to the dangerous area near the foramen on the roof of the fourth ventricle. Dr. Watkins said that he could not quote cases of actual harm resulting from this procedure. But people doing a lot of this work had found that they got better results from avoiding large lumbar punctures. It had been shown in animals that if organisms were injected into the blood streaem, there was no meningeal infection. If cerebro-spinal fluid was drawn off at the same time, meningitis developed. When the organisms were in the blood stream, as in lateral sinus thrombosis, organisms might enter the subarachnoid space and produce a meningitis if massive puncture were performed.

Dr. Halloran had suggested that he (Dr. Watkins) thought that Stewart's results were so bad that they pointed to operation being useless. Dr. Watkins did not look at it quite in that way. He thought that in future, when they had a better understanding of the condition, results would be much better. Part of the criticism he accepted. In three out of the eight instances where the patient had recovered, organisms were grown on culture. Dr. Watkins had been through many (about 230) case records, and in his opinion this percentage of recoveries was much higher than the percentage usually obtained when positive cultures had been obtained.

It was difficult to compare the results of these surgeons, because some would call the condition meningitis where others would call it meningismus with increased cell content.

Dr. Anderson, in reply, thanked Professor Dawson for his comment and for telling of the experimental work which confirmed the contention raised that the infection of meningitis was in the major part through the blood stream rather than local.

Dr. Mills had referred to increase in the cerebro-spinal fluid, which was due either to an increase of fluid or to decreased absorption. The question of hydrocephalus occupied the attention of many physicians and must be considered from both aspects. Undoubtedly there were

cases in which there was over-secretion from over-action of the plexus resulting in villus hypertrophy. On the other hand, it might be due to a decrease in the absorption of the cerebro-spinal fluid due to an alteration in the arachnoidal villi and arachnoid adhesions.

Dr. Anderson was glad to hear Dr. Tebbutt deny that the pathologist exulted over the finding of the pneumococcus. Dr. Anderson also referred to three cases of meningitis due to the influenza bacillus, which had come under her notice during the last three weeks. Serum had been administered at the Royal Alexandra Hospital for Children, but in all instances it had failed.

In regard to accessory spaces and the increase of cerebro-spinal fluid under altered conditions, Brain and Strauss had given figures in "Recent Advances in Neurology" to show that nine-tenths of the cerebro-spinal fluid was absorbed above the level of the tentorium and that only under special conditions did the spaces below come into play.

In regard to inflammatory cells, Dr. Anderson spoke of the importance of infection of the plexus as the first factor in generalized meningitis and as being the first source of inflammatory cells. The small area of the chorioid plexus could not possibly be responsible alone for the enormous amount of exudate.

In the treatment of the meningococcus, serum was possibly manufactured from cultures not obtained from Australian patients, and it was perhaps for this reason not always efficacious. Dr. Anderson referred to several cases at the Royal Alexandra Hospital for Children in which the serum was ineffectual. The cultures obtained did not appear to correspond entirely with those of workers in other parts of the world.

Dr. O. Latham, in reply to Dr. Mills's question as to why in *encephalitis lethargica* and other similar diseases the cerebro-spinal fluid was so plentiful and why so much œdema of the brain was present, suggested that, inasmuch as in these diseases and general paralysis of the insane the vascular system seemed to bear the brunt of the attack (especially was this so of the smaller blood vessels and capillaries), attention should be directed to this system. That the smaller vessels became progressively more and more impervious was evident from the numbers of new capillaries constantly being formed. Now the blood had to reach the veins to avoid stasis. But this passage was impeded at the capillary barrier. Again, the choking of the perivascular lymph channels by hosts of mononuclear cells furthermore impeded the brain lymph already exuded from the vessels. This fluid had to pass along these channels to the arachnoid, there to join up with the cerebro-spinal fluid secreted by the chorioid plexus, which had a slightly different composition. Finally, in high intracranial pressure these perivascular spaces were obviously dilated and the flow of their contents impeded. Dr. Latham had just examined a cerebral glioma, a rare form spreading by the leptomeninges, which had actually invaded these dilated spaces from the arachnoid system. This direction of travel was possible for other particles too.

MEDICO-POLITICAL.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on October 31, 1931, DR. GEORGE BELL, the President, in the chair.

The Metropolitan Hospitals Contribution Fund of New South Wales.

DR. GEORGE BELL explained that the meeting had been called for the purpose of considering the proposed principles of the Metropolitan Hospitals Contribution Fund of New South Wales, which had been drawn up by the Hospitals Commission of New South Wales.

DR. R. B. WADE explained the proposed principles at some length. The principles are as follow.

Name: The name of the fund shall be the Metropolitan Hospitals Contribution Fund of New South Wales.

Control: The control of the fund shall be vested in a council constituted by the participating hospitals and the Hospital Saturday Fund. The Council shall make such by-laws and rules as are necessary for the organizing and management of the fund.

Representation on the Council shall be on the following basis:

Hospitals with 301 or more available beds, three representatives each.
Hospitals with 201 to 300 available beds, two representatives each.
Hospitals with under 201 available beds, one representative each.
The Hospital Saturday Fund, three representatives.
The Hospitals Commission of New South Wales, one representative.

There shall be an executive committee of 15 persons elected annually by and from the members of the Council, but there shall not be more than one representative of any hospital or of the Hospital Saturday Fund on the executive.

Election of the executive shall be on a group basis, as follows, each group electing its own member or members by preferential voting.

	Members.
Royal Prince Alfred, Sydney, Saint Vincent's, Royal Alexandra for Children	4
Royal North Shore, Manly, Mater Misericordiæ	2
Western Suburbs, Lewisham, Balmain, Marrickville	2
Royal South Sydney, St. George, Canterbury, Auburn, Parramatta, Saint Joseph's	1
Crown Street Women's, Rachel Forster, Saint Margaret's, New South Wales Community Hospital (Langton Clinic)	2
Benevolent Society of New South Wales	1
The Hospital Saturday Fund	1
The Hospitals Commission of New South Wales	1

Objects: The objects of the fund shall be:

(*a*) To conduct a scheme of systematic contributions in the metropolitan area to provide a regular volume of income for the participating hospitals.

(*b*) To afford the benefits hereafter outlined to the contributors and their dependants.

Rate of Contributions: The rate of contribution shall be sixpence per week from every person willing to join the fund.

Benefits to Contributors:

(*a*) Contributors who, by reason of their financial circumstances, are eligible for admission to a public hospital and are medically certified as requiring indoor accommodation in an acute hospital, shall not be required to make any payments to the hospital towards the cost of hospitalization of themselves or their dependants and shall not be required to pay any medical fees.

(*b*) Contributors who, by reason of their financial circumstances at the time, are admitted to a hospital on a recommendation on Form 4 or Form 5 under the *Public Hospitals Act*, shall be given credit of 7s. *per diem* off the cost of accommodation for private or intermediate patients, as the case may be, and shall only be required to pay the hospital the difference between 7s. *per diem* and the charges fixed for private or intermediate accommodation and the medical fees as prescribed by the regulations under the *Public Hospitals Act* for private and intermediate patients.

(*c*) Contributors admitted to any hospital not included in the second or third schedule to the *Public Hospitals Act*, shall have the right to claim on the prescribed form, supported by receipted accounts for the hospital charges paid and by the certificate of the attending medical officer, reimbursement of the charges of such hospital to the extent of 6s. *per diem* for a period not exceeding eight weeks in any one year.

Contributors will become entitled to benefits after being financial for two months, except in the case of accidents in which benefits become immediately available, and of obstetric patients, who will be required to be financial for at least six months during the first twelve months of the operation of this fund, and thereafter the period shall be nine months.

Benefits shall apply to contributors and dependants admitted to an acute hospital for a period of twelve weeks in any given twelve months, after which the provisions of Section 30 of the Act and the regulations and by-laws thereunder will apply.

Contributors who, as members of any society or organization, are entitled to hospital payment therefrom, shall (*a*) in the case of private and intermediate patients have the amount credited towards the additional cost of such private or intermediate accommodation, and (*b*) in the case of other patients have the amount credited to their contribution account.

In the event of the temporary absence of a contributor from the metropolitan area, and of his admission to a public hospital owing to accident or other emergency, the fund will pay such hospital at the rate of 6s. *per diem* for a period not exceeding eight weeks in any one year.

On the discharge of a contributor who, by reason of his financial circumstances, has been a patient in a public ward, he shall be entitled to such after-treatment as is necessary in the outdoor department of the hospital without charge for one month.

Contributors admitted to the public wards of a hospital shall receive, without extra charge, the whole of the services of the hospital.

Contributors shall be entitled, where medically certified to be necessary, to X ray and other scientific services by a participating hospital as follows.

(*a*) Patients eligible for public ward accommodation without extra charge.

(*b*) Private and intermediate patients, at such charges to the hospital as shall be determined, together with the fees of the radiologist, pathologist *et cetera*, as the case may be, but no such examination or investigation shall be made except by written request of a medical practitioner, to whom the reports *et cetera* shall be forwarded.

Organization: For the purposes of organization of contributors, the area in which the scheme operates shall be divided into sections and subsections, each of which shall work under the direction of the central office.

The City of Sydney may comprise a number of sections and subsections.

The organization of sections may, and of subsections shall, be on the basis of voluntary support and assistance.

All administrative and other expenses of the section shall be paid by the Central Office.

The fund shall not organize any entertainment, fête or other movement for the purpose of raising funds; this does not prevent the fund from receiving, holding and distributing any gifts, bequests, donations or the like for the benefit of any or all of the hospitals in the area.

The whole of the income of the fund after payment of all administrative and other prescribed charges shall be apportioned to the participating hospitals on the basis of work done and be paid by monthly advances with half-yearly adjustments, and consideration shall be given to the number of contributors treated during the period.

General: The operation of the fund shall not interfere with the ordinary sources of income of the participating hospitals in so far as such sources do not include contribution schemes with hospital benefits.

Each participant shall undertake to cooperate in every way possible with the executive in furthering the scheme.

The hospitals shall not give any form of preference to any person because of his being a contributor.

A contributor of less than twelve months' standing who becomes a patient and on his discharge ceases to contribute, shall be liable to pay the hospital the difference, if any, between the total of his contribution and the amount for which he has been assessed as liable to pay the hospital, having regard to his then financial circumstances.

The executive shall arrange as far as possible for reciprocity with all public hospitals outside the metropolitan area in regard to payments on account of contributors to respective schemes.

Financial: Financial membership of the metropolitan fund shall be transferable to any fund outside the metropolitan area.

A contributor shall, in order to receive hospital benefits, be financial at the time of admission.

The executive of the fund may establish a special surgical appliance fund for the purpose of supplying destitute persons (whether contributors to the fund or not) with appliances as follows: Artificial limbs, special appliances made for the sole use of the patients, spectacles, and any instrument or appliance which, after closest consideration, the executive finds essential to the well-being of the patient.

Definitions:

"Dependant"—wife, husband and/or child under the age of 17 years, and the following where it can be shown they are entirely dependent on the contributor: Father, mother, sister or brother (under the age of 17 years).

"Contributor," where used, shall be read as including dependants.

"Metropolitan area" means the area embracing the districts immediately served by the participating hospitals.

"Hospital" means public hospital in the metropolitan area unless otherwise specified.

"Financial" means not in arrears for more than two months.

After a long discussion, in which many members took part, the following resolutions were adopted:

1. With regard to the Metropolitan Hospitals Contribution Fund, the medical profession affirms its traditional principle of free treatment of the indigent sick, but insists that its interests be conserved with regard to other classes of the community by payment for services rendered, and that this principle be recognized in all contributory schemes providing hospital and medical services to the community.

2. That this meeting is prepared to support the Metropolitan Hospitals Contribution scheme provided that proper arrangements are made to safeguard the interests of medical practitioners in regard to intermediate and private patients and that these interests may be safeguarded:

   (*a*) By establishing community services in all metropolitan hospitals;

   (*b*) By provision of a uniform system of classification, acceptable to the medical profession, of the different classes of patients—public, intermediate and private, according to their circumstances;

   (*c*) By establishing machinery whereby the medical practitioner in attendance shall be able to charge and recover fees.

3. That this meeting is of the opinion that any contribution scheme, industrial or otherwise, which provides for out-patient services to the contributors, is not acceptable to members of the New South Wales Branch of the British Medical Association.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Warne, James, M.B., B.S., 1915 (Univ. Melbourne), 116, Wellington Parade, East Melbourne, C.2.

Day, Ellen J. M., M.B., B.S., 1927 (Univ. Melbourne), Whittlesea.

Hendry, William Joseph, M.B., B.S., 1929 (Univ. Melbourne), Children's Hospital, Carlton, N.3.

Wood Jones, Frederic, F.R.C.S., University, Carlton, N.3.

Medical Practice.

MEDICAL SERVICE IN VICTORIA.

THE following article has been forwarded for publication by the Council of the Victorian Branch of the British Medical Association. Reference to this contribution is made in the leading article of this issue.

With the improvement of general education of the people, the elevation of the standard of living and comfort, and the advancement of medical science, there has developed a demand for increased medical and improved institutional care in sickness.

The problem of meeting the increasing demand for public aid has become rapidly greater as the financial and economic difficulties of the world have developed since the war. A crisis has been created in the maintenance of Australian public hospitals since the fall in world prices.

The combined overdrafts of seven public hospitals in Melbourne amount to £196,000 at present, and the hospitals cannot get any further financial accommodation. Expenditure exceeds revenue. They must curtail their efforts while the state of unemployment and need for public assistance is at its highest peak, or else find some new source of income.

In England in 1911 the *National Insurance Act* was passed, providing as medical benefit domiciliary service for wage-earners. In 1921 the voluntary hospitals were faced with a financial crisis and the Cave Commission recommended the development of contributory schemes, which quickly transformed the deficit to a credit and at the same time converted these voluntary charities into public utilities with honorary medical service. In 1929 the *Local Government Act* was passed, which transferred the control of poor law hospitals to the municipalities. Thus there is developing in England a system of 150,000 hospital beds with hospital staffs, salaried or honorary, to render nursing and hospital service to 85% of the people, and a domiciliary medical service for 15,000,000 breadwinners paid for on a fixed capitation basis.

To relieve the position of public hospitals in Australia, contributory schemes are being initiated. For sixpence a week contributors are promised public hospital treatment for themselves and their dependants when hospital treatment is necessary, unless they can afford to pay medical fees. At least 60% of the people are alert to enjoy the advantages of such economy. Their ability to pay medical fees is difficult to establish or dispute. So, in increasing numbers the public will enjoy the privilege of free medical service or comprehensive service for sixpence a week—a service which they are assured of, and by inference have a right to, being "unable" to pay medical fees. The hospitals accepting money for service are to contributors no longer charities, but public utilities. The contributor is relieved of indignancy and obligation, and offered service, including medical service, for sixpence a week for himself and dependants. What makes the scheme so popular to everybody except the medical profession is that the hospitals sell nursing and medical service for sixpence a week and pay nothing for the medical service. This is paid for (i) by the thrifty independent middle classes, who struggle with higher medical fees, and (ii) by the medical profession.

The development of this system can only end in the payment for medical service by the institutions. This is a system by which, in large hospitals, the patient loses his name and becomes a number with a curious or simple disease; and by which he must take whatever fortune sends him in the way of doctors and treatment; by which neither he nor his friends have any real prerogative; and by which, for sixpence a week he is relieved of his responsibility and also of his freedom and independence.

The Victorian Branch of the British Medical Association is quite conscious of all this. It is determined that in the interests of the people this will not happen in Melbourne. It has clearly defined its position with those organizations concerned in and proposing contributory schemes. It declares that the fundamental principles of efficient medical and nursing services are individual freedom, individual independence and individual responsibility; that members of the medical profession are the chief executive officers of the public hospitals which they serve in a voluntary capacity; and that they will not agree to these hospitals being converted into public utilities accepting free medical service.

Any contributory scheme which is launched, must set aside an agreed proportion of the weekly contribution to be paid into a separate fund, such being adequate provision for consulting and specialist service. Medical benefits will thus be available to subscribers requiring hospital medical service. Such benefits will be in the form of cash benefit. Thus there is maintained for the individual patient that which, if he knew it, is his most cherished possession, his freedom and independence.

The Victorian Branch of the British Medical Association realizes the necessity for action which will maintain: (i) adequate medical service to all sections of the community, (ii) solvency and activity of public hospitals.

After mature consideration the Victorian Branch, at a special meeting held on October 26, 1931, approved of:

1. The initiation of an insurance scheme which will enable people below defined income limits to provide for:

   (*a*) Domiciliary service.
   (*b*) Investigational service.
   (*c*) Consulting and specialist service.
   (*d*) Nursing and hospital service.
   (*e*) Pharmaceutical service.

2. The scheme being one by voluntary insurance.

3. The scheme being initiated and controlled by the medical profession in respect of (*a*), (*b*) and (*e*).

4. Where a capitation fee is paid to doctors as a means of insurance for any of the above services, the patient at the same time shall be required to pay a visitation or service fee for each service rendered.

5. The establishment of funds to provide cash benefits to assist in the payment of consultant and specialist services.

6. The establishment of a fund by lay bodies to provide cash benefits to assist in the payment of nursing and hospital services in conjunction with cash benefit for consultant and specialist services.

7. Where a contributory scheme towards the cost of nursing and hospital services is initiated or under the control of lay bodies:

   (*a*) The medical profession shall have adequate representation on the executive body of such scheme.
   (*b*) A proportion of the subscriber's contribution agreed upon shall be used solely for the provision of medical benefit.
   (*c*) All benefits available shall be in the form of cash benefits to subscribers.
   (*d*) Contributors, as such, shall not be given direct representation upon the executive of such scheme.

8. The Council taking what action it considers necessary to promote contributory schemes in accordance with the foregoing resolutions.

Dealing *seriatim* with these resolutions:

Resolution 1 envelops the whole of the major requirements of the people. Establishment of such a service will relieve the need for public aid.

Resolution 2 approves of a voluntary scheme, because it would not be possible at present to secure the passage of legislation for a compulsory scheme.

Resolution 3 insists that wherever possible the scheme is under medical control. Lay control of the relations between doctor and patient has always proved unsatisfactory. Part (*c*) and (*d*), however, must go together.

Resolution 4 recognizes that this system (i) lowers the necessary capitation fee, (ii) protects doctors from being harassed by trivial complaints of the importunate and allows more time to be placed at the disposal of the really sick contributors, (iii) it allows a uniform capitation fee for all districts and a readjustment of remuneration for diverse conditions of service in different districts by varying the service fee according to circumstances in the different districts.

Resolutions 5 and 6 are approved because schemes must be established from the same contribution. If 6 were established without 5, then there would be no provision at all for medical service. No provision should be made for 6 without 5. Therefore both funds will be under lay control.

Resolution 7 (*a*) is necessary to coordinate 5 and 6 with 1 (*a*), (*b*) and (*c*).

(*b*) insists that an agreed proportion of the contribution will be paid into separate funds: (*a*) nursing and hospital benefit fund, (*b*) consulting and specialist benefit fund. These funds will not be interchangeable, so that extravagance in one will not determine insolvency of the other.

(*c*) maintains free choice of doctor for the patient and freedom of contract.

(*d*) is in recognition of the fact that the fund is prone to dictate the policy of the hospitals whither the patients go. If the hospitals do not conform to the wishes of the fund, then the fund withdraws its support. If the fund is controlled by contributors, then control of hospitals falls into the hands of those who have made no effort to establish them and have had no experience. The bodies who control the fund should be mainly those who have created hospitals conducted neither for profit nor charity (that is, community hospitals of church organizations or those organizations whose stability depends on maintenance of health and who will undertake to provide capital wherewith community hospitals may be established—friendly societies and insurance companies).

The detailed scheme as presented to the public is appended.

The medical profession of Australia is like the medical profession of all other countries—at the cross-roads. It has been said "that no problem is settled until it is settled right." What is right will depend upon the citizenship of our generation. There is a great conflict of ideals and ideas between fascism and communism in the world today. The British Isles have swung at the recent general and municipal elections from socialism to regulated freedom.

The public hospitals are the greatest pacemakers of socialization in any community. Contributory schemes as in England and Sydney, as announced in the Sydney Press, prosper its progress. The policy of the Victorian Branch of the British Medical Association stands for regulated freedom, independence and individual responsibility.

Its successful future depends not upon a reactionary spirit, but upon the professional earnestness, loyalty and citizenship of the Victorian medical profession.

SYNOPSIS OF SCHEMES.

Part I: (*a*) Nursing and hospital service, (*b*) consulting and specialist service.

Part II: (*a*) Domiciliary service, (*b*) investigational service, (*c*) pharmaceutical.

Part I.

Contributions: Nursing and hospital service, consulting and specialist service, 9d. per week, 9s. 9d. per quarter, 39s. per year.

Cash Benefits to Subscribers: (i) Hospital expenses, £2 per week; (ii) consultant and specialist service: surgical operation (major), up to £9; confinement, £3 3s.; anæsthetic, £1 1s.

Management of Fund: Bodies representative of: (i) voluntary organizations which have created and controlled hospitals neither for charity nor profit; (ii) voluntary cooperative organizations whose solvency depends upon maintenance of health—friendly societies, commercial insurance companies; (iii) British Medical Association; (iv) Australasian Trained Nurses' Association.

Hospital Bed Provision: Establishment of bureau.

Part II.

Management: Board of five nominated by the Council of the Victorian Branch of the British Medical Association.

Contribution: Domiciliary service, investigational service, pharmaceutical service: A weekly rate of 5d. single male or female under 21 years, 8d. subscriber without dependants, 9d. subscriber and wife and no child, 10d. subscriber and wife and one child, 10d. subscriber and wife and two children, 1s. subscriber and wife and more than two children, *plus* a small charge for each service.

Income Limits: Below £208 *per annum* for single man or woman; below £312 *per annum* for married man, with an additional allowance of £25 for each child.

Subscribers' Service Charges: Domiciliary service, 3s. 6d. or other sum agreed upon; investigational service, 5s. or price of X ray films; pharmaceutical service, approximately half present private prescription rate.

Board will insure that highest professional standards are maintained.

Schemes to be initiated experimentally first in selected district.

Mileage in excess of two miles' radius of doctor's residence to be determined and charged.

Emergency treatment: Subscribers to have right to call in another panel doctor upon failure of his own doctor to attend after reasonable notice.

Appointment of investigating officers to examine applicants for public hospital aid.

Protection of scheme against exploitation.

Obituary.

PHILIP ALOYSIUS PARER.

DR. PHILIP ALOYSIUS PARER, who died at Kew, Victoria, on October 15, 1931, was born at Fitzroy, Victoria, in 1882. He was educated at the Christian Brothers' College, East Melbourne, and at Saint Patrick's College. He studied medicine at the University of Melbourne and graduated Bachelor of Medicine and Bachelor of Surgery in 1911. After graduation he served as resident medical officer at Warrnambool Hospital and the Children's Hospital, Carlton. In 1913 he started practice in Fitzroy and by close attention to his work and his knowledge of men he built up a large practice. Six years ago he had to give up his practice on account of ill health. He went to live in Kew, where he devoted most of his attention to his garden and to his aviary, for he was a lover of birds. He is survived by his wife and two daughters.

Dr. J. Forbes Mackenzie writes:

Phil. Parer died much too early. He belonged to that type of general practitioner who, when one comes to think of it, does the bulk of the doctoring for the community. The type of man who, when tired out by a wearying night of obstetrical worry, has to turn round and make an accurate diagnosis between a pancreatitis requiring immediate operation, and the exaggerated moaning groan of a "clubby" who is intent on getting something out of his doctor for all the years he has been paying into the lodge. I have known Parer ever since he started practice in Fitzroy, in a poor locality, where, by sheer hard work, he built up a tremendous practice. His unfailing good humour and calm efficiency endeared him to his patients, and indeed to everyone who knew him. How well he could laugh off the moans of the moaner, reassure him and never get angry with him; and then the trouble he would go to at all hours to alleviate the sufferings of some poor hopeless case, where there was no remote prospect of payment.

This man was a good doctor in every sense of the word; and an apprenticeship of six months to one of his quality would be a wonderful educational finish for any young graduate. Pylephlebitis following the recrudescence of a diverticular abscess was responsible for the ending up of a most useful life. Dr. Parer leaves a widow and two daughters to mourn his loss, and to them our deepest sympathy is extended.

Dr. W. J. Newing writes:

The untimely death of Philip Parer will be regretted by an unusually large number of friends who were

attracted by his very pleasing personality. The calls of a large general practice gave him little time for outside pursuits, but did not prevent him always taking a most active part in the welfare of the General Practitioners' Section.

Possessed of a wide experience of human nature, his advice was always tempered with wisdom, and his hints on difficult midwifery, of which he was a master, have proved invaluable to many younger men.

LESLIE OSWALD SHERIDAN POIDEVIN.

WE regret to announce the death of Dr. Leslie Oswald Sheridan Poidevin, which occurred on November 19, 1931, at Sydney, New South Wales.

Post-Graduate Work.

POST-GRADUATE LECTURES IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee announces that Mr. C. H. Fagge, Senior Surgeon, Guy's Hospital, London, who is coming to Australia to be present at the annual meeting of the Royal Australasian College of Surgeons, has consented to give a series of lectures on surgical subjects of interest to general practitioners during his stay in Melbourne. The lectures will be delivered at the Medical Society Hall, Albert Street, East Melbourne, on the evening of February 11, 12 and 15, 1932. The fee for the course will be two guineas. The titles of the lectures and further details will be announced later.

Correspondence.

WORKERS' COMPENSATION: TYPHOID FEVER.

SIR: One of the most important problems that confront the student of infectious diseases is the elucidation of the means by which the disease spreads. Any light thrown on this point is at all times welcome.

It will, I think, be accepted as a matter of congratulation that we have in this State a body which from time to time feels itself in a position to decide some aspects of the problem.

A recent decision has cleared up a somewhat obscure question as to how typhoid spreads in this city. Our experience has been that, apart from small localized epidemics definitely traced to some food infection, the very large majority of cases occur as isolated incidents with no apparent connexion between them or with any known source of infection. Many years ago it was thought, on what were regarded as sufficient grounds then, that there was an essential connexion between the system of conservancy in a community and the incidence of the disease. As the water carried system of sewerage extended, so the number of cases fell off, and this was considered as confirmation of this view. But during the same period other diseases (tuberculosis *et cetera*) have fallen off, and no claim such as this has been made or can be supported in this connexion. Also it is found that there is no greater incidence of typhoid in houses not connected with the sewer than in those sewered. Therefore, this theory, although strongly rooted in popular and even official minds, loses much, if not all, of its weight. There must be some other factor operating, and students of epidemiology have been racking their brains to discover the missing link. The existence of carriers, so well established now, affords a ready explanation of the means whereby the infection can be spread, but this does not complete the chain, and something more is wanted to show how the infection is conveyed from a carrier to a potential patient. All modern researches go to show that the typhoid organism has a very short life outside the human body, especially when it is living in competition with other organisms. Therefore, the danger from these organisms, when they have been passed from the human body, rapidly disappears. The attraction of dejecta for flies is well known, and it has been shown that the typhoid organisms will live for some days on and in the bodies of flies. In ordinary households it is found that flies mostly frequent lavatories and kitchens. Applying this knowledge, it does not require any great flight of imagination to see the opportunities for the conveyance of infected matter from the point where it has left the human body to a point where it has a chance of being ingested by other persons. Furthermore, in this translation, if the organisms are deposited in or on foodstuffs suitable for their growth, such as milk, cold meat *et cetera*, the unfortunate person who consumes them, will not receive a few organisms dropped by a fly, but a heavy dose of a growing culture. The person swallowing the dose may not develop the disease on account of insusceptibility or because the dose is too small or the virulence of the organism too low. This, of course, is fortunate, because, owing to the wide spread of the organism throughout the community and the impracticability of any individual in ordinary life avoiding the accidental ingestion of the organisms from time to time, we must all have swallowed them. A sewer worker is not exempt from this risk.

The decision referred to above was given in the case of a man who worked in the reconstruction of a sewer in which he came in contact with fæcal matter. He developed typhoid. It was decided that he must have been infected from this fæcal matter. This gets over all our difficulties. It is so beautifully simple that one can almost see the well known "Don't Argue" advertisement and feel the protesting hand approaching.

Curiosity to learn a few details of how it all happened elicited the following theories, built up from and designed to show the connexion between the two major premises stated above. The first theory is that the sewage must have contained typhoid organisms. This is based on a general assumption and also because organisms indistinguisable from typhoid bacilli had been recently described as being found in Belfast sewage. It is to be noted that this was Belfast, not Sydney, sewage and that there was nothing to show that the organisms found were sufficiently virulent to give rise to the disease. However, this theory was evidently held to show that the sewage with which the man came in contact contained organisms capable of giving him the disease. The next theory required was to provide evidence of the source of the organisms. As it was accepted that the organisms must have come from some person, it was necessary to assume the existence of some undetected case or carrier within the area served by the sewer. There had been no recognized cases within the part. This theory seems to be quite satisfying, but, if correct, why should not the reasonable probability be that the train of events would then follow the same course and sequence as it does and has done in hundreds of other cases in this country? This consideration cannot be lightly brushed aside.

The acceptance of these theories closes two gaps, but more are required to complete the tale. How did the organisms, existing in theory in the sewage and derived from a theoretical source, find their way into the man and cause the disease. The further theory is that the man must have conveyed the organisms, which another theory again assumes to be recent and virulent, into his mouth and swallowed them. The explanation given is quite simple. A keen observer gave evidence that he saw two fellow workmen roll cigarettes and smoke them. He infers that the man in question did the same and must have thereby swallowed the theoretically infected sewage. This seems to involve a few more theories, but as so many have been accepted, a few more are apparently neither here nor there. Incidentally, this raises a rather interesting point, perhaps not quite relevant to the main issue under consideration. If a man, during the time he is supposed to be working, stops to make a cigarette, adds sewage to it from his hands contaminated by his work, removes the sewage therefrom by his mouth and swallows it, and if further it can be shown that he was infected with typhoid

by this sewage, can it be held that he received an injury arising out of and in the course of his employment? His work does not require him to smoke, or even if he imagined that he could not work without smoking, it does not require him to add sewage to his tobacco and then swallow it. If he likes this sort of thing, surely his employment should not carry the consequences of his peculiar tastes.

The man worked for thirty-five hours a week in the sewer. For the balance of the week (133 hours), it is presumed that he led the life of any ordinary citizen of his class, and there was certainly nothing to show that during the hours away from work he took any precautions to protect himself from the chances of infection to which all citizens are exposed. As a matter of practice, it is quite impossible for any individual to surround himself with an absolutely infallible wall of precautions so long as he mixes with his fellow men. He might attain security by living in a sterile test tube.

As the theories built up to the account for the phenomena observed required that he was not infected during the hours away from work, he was obviously regarded as a man not subject to the same risks as others. Why this should be so was not explained, but perhaps it was that he worked in a sewer containing sewage. Is it not open to conclude from this that work in a sewer is a protection from other risks, and that complete immunity might be attained by working in a sewer and observing the simple precaution of not swallowing any sewage?

However, as all the links in the chain have now been forged, the "balance of probabilities," blessed word, reminiscent of Mesopotamia, is now trotted out, and the point is established that if a man works in a sewer in which there is sewage, and develops typhoid, he contracts it from the sewage. So now we know.

It is satisfying to have this point settled. I have been in close contact with hundreds of men working in sewers, containing sewage, for over a quarter of a century, and have never seen a case of typhoid amongst them. My experience in this connexion is by no means unique. I have pondered frequently why this should be. On all our old theories it is quite wrong. I have been almost persuaded that it was that our theories were wrong, but now I am comforted and back in the old fold. Our old views must be right. The edict is given. These hundreds of workers in sewers, containing sewage, have been most disobliging. They were quite wrong in not having had typhoid, and very inconsiderate in upsetting us all in this way.

Yours, etc.,

E. S. STOKES.

341, Pitt Street,
Sydney,
November 9, 1931.

BINIODIDE OF MERCURY.

SIR: For ten years I have been using biniodide of mercury solution, one in one thousand, in the manner indicated by "Physician." Its application in wounds sustained in the use of farm machinery and in road accidents has invariably been associated with healing by first intention.

Yours, etc.,

F. J. BURNS.

Ganmain,
New South Wales,
November 11, 1931.

MUSCULAR EXERCISE AND GASTRO-INTESTINAL PERISTALSIS.

SIR: In your issue of November 7 Dr. C. E. Corlette expresses the opinion that it is improbable that muscular exercise increases gastro-intestinal peristalsis. As the question has been shown to be of medico-legal importance it is to be hoped that a radiographer will at once decide it by direct experiment, if this has not been done already. In the meantime certain physiological considerations make Dr. Corlette's view extremely probable: (i) During exercise the very great increase of the blood flow through the active muscles is made possible by a great diminution of that through the splanchnic area. It is most unlikely that increased rather than decreased peristalsis would accompany a greatly lessened blood supply to the gastro-intestinal muscles. (ii) During exercise there is a considerable outpouring of adrenalin. Injection of adrenalin inhibits gastro-intestinal peristalsis. (iii) The carbonic and lactic acids added to the circulation during exercise would possibly diminish the activity of the gastro-intestinal musculature.

Yours, etc.,

ERIC JEFFREY.

UNTRAINED NURSES.

SIR: I have been directed by my Council and with the consent of the Australian Nursing Federation to forward you a copy of a letter received from the latter body and to ask if you would be good enough to insert it in a future issue of your journal.

Yours, etc.,

C. STANTON CROUCH,
Secretary.

Medical Society Hall,
Albert Street,
East Melbourne,
November 11, 1931.

AUSTRALIAN NURSING FEDERATION.
(Royal Victorian Trained Nurses' Association.)
Colonial Mutual Building,
314, Collins Street,
Melbourne, C.1,
14th July, 1931.

Secretary,
British Medical Association,
Albert Street,
East Melbourne.

Dear Sir,

Recently a member of the medical profession brought before the notice of my Council the fact that there is at the present time a considerable increase in the number of untrained women who are posing as trained nurses, and inquiry was made as to whether any action could be taken by this Association to prevent this practice.

The inquirer was informed that some time ago, when a bill to amend the *Nurses' Act* was before Parliament, this Association approached the Minister of Health asking for the insertion of a clause in the *Nurses' Act* for the further protection of the general trained nurse. This, however, was not included in the bill, but it is the intention of my Council, when a Nurses' Bill is being drafted, to again bring this request before the Minister of Health.

My Council has directed me to bring this matter before the notice of the British Medical Association, asking for its support and cooperation in the endeavour to put an end to this practice.

Trusting that this matter will receive the sympathetic support of your members.

I am,
Yours faithfully,
(*Signed*) M. ANDERSON,
Secretary.

TRAUMA AND THE HEART.

SIR: As Dr. C. E. Corlette rightly pointed out the great importance of a true conception of the relation of trauma to the heart and viscera in compensation cases, one may be pardoned for doubting if the last word has

been said on the matter or that Dr. Smith has established his thesis. We are asked to believe the healthy heart cannot be injured by exertion, however severe, without any accurate definition of what is meant by "health" or "healthy." All I can gather is that a workman with no declared signs or symptoms of heart disease has a "healthy" heart and is not subject to the general practitioner's favourite certificate of "heart strain." Yet, since we have the horrible example of such a one put through a clinical fourth degree after signing such a certificate, it is logical to declare that until the "healthy" heart is so defined, the practitioner was as much entitled to speak of traumatic heart as the specialist to deny its presence. A very simple physical test would have decided the question in a few minutes, whereas curing an alleged condition by suggestion proves nothing except that it may have been real enough at first, but was cured when the suggestion was made.

Cases which are certified by the attendant as traumatic and as strongly denied by the specialist who examines for the employer or insuring authority, are seen with different eyes. If we presume that every person, no matter what his functional activity, physique or habits may be, is a "healthy" man as long as he can do his work in a proper manner, the question is an easy one to answer. But there has unhappily grown up on the part of the specialist a tendency to discuss man in terms of the modern laboratory, in spite of the fact that humanity is not a product of the laboratory, but the latter has been produced by man and therefore is subject to all human errors. You cannot reduce man to a formula or apply mathematical principles to humanity. Dr. Hans Much, of Munich, in "*Moderne Biologie*" (*Heft ii*), actually denies biology the status of a science, since it cannot be reduced to mathematical principles! So we are on safe ground in denying the truth of scientific statements if they are contrary to experience or experiment in medicine, even though we cannot explain the facts ourselves. Now if one is called to attend a man who, in the course of his work has been submitted to unusual exertion followed by pain under the sternum and who is more or less gasping like a gorged frog and who on recovery suffers the same thing if untreated each time he over-exerts himself, one is entitled to become a pragmatist. If the heart responds to the same treatment as the strained voluntary muscle of the footballer, he is entitled to speak of heart "strain"; and to seek for some other explanation than the often contradictory experiments of the laboratories. For there is not the slightest doubt that complete and absolute rest is the first need in a spread-eagled position, followed by the Ochsner-Sherren treatment used for appendicitis. If the heart is "healthy," the ratio of pulse, respiration and temperature will be normal and constant under the test of exertion, but if "unhealthy," the ratio will be altered. And it will be found, if these men are investigated during and immediately after their work, that after a severe "strain" the "healthy" heart of rest is changed into the "unhealthy" heart of work when excessive.

Here are some extreme cases of the condition which in its minor states constitute, in my judgement, heart "strain." They were all normal people and were examined after death to verify the opinion formed that they were healthy people with healthy hearts according to the best methods available.

1. A retired man of sixty-three takes his usual walk up a hill and, while jumping from one log to another, falls down dead.

2. A man of fifty-five, with moderate blood pressure, drops dead while hitting a golf ball.

3. A farmer of forty-three, on a trying hot day at sports, helped in a tug-of-war and dropped dead when it was over.

4. A woman of thirty, in labour, attended by a midwife, got an impacted fœtal head, and during one long pain died of heart failure.

5. A native, running to get help, trips over a log and dies.

No one can object to these cases being classed as due to trauma, and whether we use the word "shock" or "strain" does not matter as long as we remember it is a distinct clinical entity. There is no need to think of "strain" as the complement of "stress," but with its ordinary English meaning of unexpected or sudden expenditure of energy by an organ not fitted to undergo it. All men with experience in working class practices meet the condition and soon learn there is a breaking point for these people, varying with age, occupation, habits and physique. A blacksmith, for example, in one case found he could lift a plough with ease, but any attempt to lift a scoop brought on a "clutch in his chest." One week after he had been passed as a good life for insurance he tested the matter with the usual result.

I suggest there is a good case for the certificate of "heart strain" in spite of the absence of arrhythmia, hyperpiesis or classical symptoms of circulatory trouble, that the clinical picture is definite, the diagnosis usually easy, the treatment effectual and the prognosis good. The poor devil who so often turns up in our surgeries with a history of collapse, either partial or complete, after some unusual exertion in his work, will get a better chance if we recognize his condition as genuine instead of treating him as a malingerer or subject to a "neurosis."

Yours, etc.,

J. F. MERRILLEES.

Alexandra,
Victoria,
November 13, 1931.

TONSILLITIS.

SIR: My experience extends over forty years and has been in a much wider field than that of Dr. Graham Brown. I agree that dissection (diathermy excepted) is the only satisfactory method. From 1917 to 1926 it was my universal practice.

I admit that diathermy is tedious. Why should an impatient person like myself abandon a perfectly satisfactory technique for a more laborious task? Entirely in the interest of my patients. Because (i) it is perfectly safe, (ii) it is free from hæmorrhage, (iii) it is free from pain, and (iv) there is no invalidity. All operations conducted without skill will give bad results, and though I advocate that skilled and specialists are the safest people to deal with tonsils, we cannot close our eyes to the fact that nearly everyone considers the tonsil fair game. As a universal method diathermy will do less harm than enucleation.

Dr. Graham Brown confesses he has no personal experience of treating tonsils with diathermy, by making the statement about "bottling up sepsis by scarring over." His experience with the diathermy knife, however, should have taught him of its impossibility. Has he such a bad case to handle that he should indulge in abuse of the other side?

I have only met two patients to whom the operation was "nerve racking," and as they jumped when the spatula was applied and could not be pacified, I did not persevere.

All his indictments against diathermy could be multiplied a thousandfold against the ordinary operation. I have never heard of a death or severe hæmorrhage from diathermy in Australia. How many have occurred from the ordinary operation during the past ten years?

Dr. Dan Mackenzie's experience, like mine, is limited to five years. Surgeons in America have been using it freely for twice that period.

As regards the lower pole and the lingual tonsil, there is no comparison as to its efficacy and ease of application.

Five years ago I would have agreed as to the necessity of removing "the tonsil, the whole tonsil and nothing but the tonsil." Today I am convinced that in adults there is no disadvantage in leaving remnants. With a little skill and patience it is quite easy to remove the lot and nothing but the tonsil. Far easier and more truly than with the most careful dissection. A small portion left after dissection is always a menace. A much larger portion left after diathermy is in my experience harmless.

In five years two patients have returned with recurring or fresh sepsis of tonsil remnants—one extra sitting was quite enough.

I would like a man of Dr. Graham Brown's great skill and wide experience to give diathermy a fair trial. I am convinced he would be converted and be just as enthusiastic as I am myself.

The machine must be inspected regularly and kept in thorough working order.

Yours, etc.,
W. KENT HUGHES.

22, Collins Street,
Melbourne,
November 13, 1931.

Books Received.

A POCKET ATLAS AND TEXT-BOOK OF THE FUNDUS OCULI, by G. L. Johnson, M.A., B.C., M.D., F.R.C.S.; Second Edition; 1931. London: Adlard and Son. Crown 8vo., pp. 223, with numerous illustrations in the text by the author and fifty-four coloured drawings selected from the author's cases by A. W. Head, F.Z.S.

Diary for the Month.

DEC. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
DEC. 1.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 2.—Victorian Branch, B.M.A.: Council.
DEC. 2.—Victorian Branch, B.M.A.: Annual General Meeting.
DEC. 3.—South Australian Branch, B.M.A.: Council.
DEC. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 10.—New South Wales Branch, B.M.A.: Branch.
DEC. 11.—Queensland Branch, B.M.A.: Branch (Annual).
DEC. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC 18.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. J. W. Rollison (B.M.A.) has been appointed Deputy Superintendent, Northfield Mental Hospital, and Medical Officer, Yatala Labour Prison; also Medical Officer, Consumptive Home, Division 2, South Australia.

* * *

Dr. C. H. J. Ramsbottom (B.M.A.) has been appointed Medical Superintendent, Benford Park Sanatorium, and Honorary Medical Officer, T.B. (*sic*) Clinic; also Consultant Visiting Medical Officer, Consumptive Home, South Australia.

* * *

Dr. W. Muir has been appointed Quarantine Officer, Esperance, Western Australia, pursuant to the provisions of the *Quarantine Act*, 1908-1924.

* * *

Dr. G. F. Lumley (B.M.A.) has been appointed Quarantine Officer pursuant to the provisions of the *Quarantine Act*, 1908-1924.

* * *

Dr. M. Schneider (B.M.A.) has been appointed Honorary Clinical Assistant to the Ophthalmic Section at the Adelaide Hospital, South Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

CITY OF BENDIGO, VICTORIA: Medical Officer of Health.
MANLY DISTRICT HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.
ROYAL ARMY MEDICAL CORPS: First Commissions.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.
SWAN HILL DISTRICT HOSPITAL, VICTORIA: Resident Medical Officer.
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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